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Part XI

# Environmental Protection Agency

## Water Quality Criteria; Availability

## ENVIRONMENTAL PROTECTION AGENCY

[FRL 1300-4]

### Water Quality Criteria; Availability

**AGENCY:** Environmental Protection Agency.

**ACTION:** Notice.

**SUMMARY:** EPA is announcing the availability for public comment of water quality criteria for the last 12 of the 65 pollutants listed as toxic under the Clean Water Act (CWA). When published in final after public comment, these water quality criteria may form the basis for enforceable standards. The criteria were developed pursuant to section 304 of the CWA and in compliance with a court order. Summaries of both aquatic-based and health-based criteria and the criteria formulation sections of the documents are published below.

**DATES:** Written comments should be submitted to the person listed below by December 31, 1979.

**FOR FURTHER INFORMATION CONTACT:** Kenneth M. Mackenthun, Director, Criteria and Standards Division (WH-585), Office of Water Planning and Standards, U.S. Environmental Protection Agency, 401 M Street, SW., Washington, D.C. 20460, telephone 202/755-0100.

**SUPPLEMENTAL INFORMATION:** EPA published 27 water quality criteria for public comment on March 15, 1979 (44 FR 15926). At that time EPA published a preamble for the criteria, a methodology for deriving aquatic life criteria, a methodology for deriving human health criteria, a summary of specific issues for commentors to address and summaries of the individual criteria documents. The information contained in the March 15 publication applies to the criteria published in this notice. EPA published an additional 26 water quality criteria for public comment on July 25, 1979 (44 FR 43660).

**AVAILABILITY OF DOCUMENTS:** Copies of the complete documents will be sent to all persons who requested copies of the initial criteria prior to the time they were published and to those who commented on the first 27 criteria. Other persons wishing to review the full documents may obtain copies from the National Technical Information Service, 5285 Port Royal Road, Springfield, Virginia (703) 557-4650. A list of the NTIS publication order numbers for all 65 criteria documents is published as an appendix to this notice. The documents also are available for public inspection and copying during normal business hours at: Public Information Reference Unit, U.S. Environmental Protection Agency, Room 2404 (rear), 401 M Street, SW., Washington, D.C. 20460. As provided in 40 CFR Part 2, a reasonable fee may be charged for copying services. Copies of these documents will also be available for review in the EPA Regional Office libraries.

Dated: September 13, 1979.

**Douglas M. Costle,**

*Administrator.*

**Appendix**

1. Acenaphthene 296 782
2. Acrolein 296 788
3. Acrylonitrile 297 915
4. Aldrin/Dieldrin 297 916
5. Antimony 296 789
6. Arsenic 292 420
7. Asbestos 297 917
8. Benzene 292 421
9. Benzidine 297 918
10. Beryllium 292 422
11. Cadmium 292 423
12. Carbon Tetrachloride 292 424
13. Chlordane 292 425
14. Chlorinated benzenes 297 919
15. Chlorinated ethanes 297 920
16. Chloroalkyl ethers 297 921
17. Chlorinated naphthalene 292 426
18. Chlorinated phenols 296 790
19. Chloroform 292 427
20. 2-chlorophenol 292 428
21. Chromium 297 922
22. Copper 296 791
23. Cyanides 296 792
24. DDT 297 923
25. Dichlorobenzenes 292 429
26. Dichlorobenzidine 296 793
27. Dichloroethylenes 292 430
28. 2,4-dichlophenol 292 431
29. Dichloropropanes/propenes 296 799
30. 2,4-dimethylphenol 292 432
31. Dinitrotoluene 296 794
32. Diphenylhydrazine 296 795
33. Endosulfan 296 783
34. Endrin 296 785
35. Ethylbenzene 296 784
36. Fluoranthene 292 433
37. Haloethers 296 796
38. Halomethanes 296 797
39. Heptachlor 292 434
40. Hexachlorobutadiene 292 435
41. Hexachlorocyclohexane 297 924
42. Hexachlorocyclopentadiene 292 436
43. Isophorone 296 798
44. Lead 292 437
45. Mercury 297 925
46. Naphthalene 296 786
47. Nickel 296 800
48. Nitrobenzene 296 801
49. Nitrophenols 296 802
50. Nitrosamines 292 438
51. Pentachlorophenol 292 439
52. Phenol 296 787
53. Phthalate esters 296 804
54. Polychlorinated biphenyls (PCBs) 296 803
55. Polynuclear aromatic hydrocarbons 297 926
56. Selenium 292 440
57. Silver 292 441
58. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) 292 442
59. Tetrachloroethylene 292 443
60. Thallium 292 444
61. Toluene 296 805
62. Toxaphene 296 806
63. Trichloroethylene 292 445
64. Vinyl chloride 292 446
65. Zinc 296 807

### Acrylonitrile

*Criteria Summary*

*Freshwater Aquatic Life.* For acrylonitrile the criterion to protect freshwater aquatic life as derived using the Guidelines is 130 μg/l as a 24-hour average and the concentration should not exceed 300 μg/l at any time.

*Saltwater Aquatic Life.* For acrylonitrile the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 130 μg/l as a 24-hour average and the concentration should not exceed 290 μg/l at any time.

*Human Health.* For the maximum protection of human health from the potential carcinogenic effects of exposure to acrylonitrile through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of acrylonitrile estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion Formulation section of this document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria of 0.08 μg/l, 0.008 μg/l, and 0.0008 μg/l, respectively. If water alone is consumed, the water concentration should be less than 0.16 μg/l to keep the lifetime cancer risk below $10^{-5}$.

*Basis for the Criteria*

*Freshwater Aquatic Life.* The maximum concentration of acrylonitrile is the Final Acute Value of 300 μg/l and the 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For acrylonitrile the criterion to protect freshwater aquatic life as derived using the Guidelines is 130 μg/l as a 24-hour average and the concentration should not exceed 300 μg/l at any time.

*Summary of Available Data*

The concentrations below have been rounded to two significant figures.

Final Fish Acute Value = 2,500 μg/l
Final Invertebrate Acute Value = 300 μg/l
Final Acute Value = 300 μg/l
Final Fish Chronic Value = not available

Final Invertebrate Chronic Value=greater than 710 μg/l
Final Plant Value=not available
Residue Limited Toxicant Concentration=not available
Final Chronic Value=greater than 710 μg/l
0.44×Final Acute Value=130 μg/l

*Saltwater Aquatic Life.* No saltwater criterion can be derived for acrylonitrile using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available.

However, results obtained with acrylonitrile and freshwater organisms indicate how a criterion may be derived.

For acrylonitrile and freshwater organisms, 0.44 times the Final Acute Value is less than the Final Chronic Value that is derived from results of a life cycle test with *Daphnia magna*. Therefore, it seems reasonable to estimate a criterion for acrylonitrile and saltwater organisms using 0.44 times the Final Acute Value. Both a Final Fish Acute Value and a Final Invertebrate Acute Value are available for acrylonitrile and freshwater organisms, and the Final Acute Value is based on the invertebrate value since it is the lower of the two. For saltwater organisms, only a Final Fish Acute Value is available. For freshwater organisms, the Final Invertebrate Acute Value divided by the Final Fish Acute Value is 300÷2,500 μg/l=0.12. Multiplying this value times the saltwater Final Fish Acute Value for acrylonitrile results in an estimated saltwater Final Invertebrate Acute Value of 0.12×2,400 μg/l=290 μg/l. Thus the estimated Final Acute Value for acrylonitrile is 290 μg/l. Multiplying the final Acute Value of 290 μg/l by 0.44 gives 130 μg/l.

The maximum concentration of acrylonitrile is the Final Acute Value of 290 μg/l, and the 24-hour average concentration is 0.44 times the final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For acrylonitrile the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 130 μg/l as a 24-hour average and the concentration should not exceed 290 μg/l at any time.

*Summary of Available Data*

The concentrations below have been rounded to two significant figures.

Final Fish Acute Value=2,400 μg/l
Final Invertebrate Acute Value=not available
Final Acute Value=2,400 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=not available
Residue Limited Toxicant Concentration=not available
Final Chronic Value=not available
0.44×Final Acute value=1100 μg/l

*Human Health.* The animal carcinogenicity studies of Norris (1977), Quast, et al. (1977), and Maltoni, et al. (1977) and the epidemiological studies of O'Berg (1977) and Monson (1977) were considered to be the most pertinent data for the determination of a water quality criterion for the protection of human health. Although the epidemiological studies showed excesses of various cancers in man, neither study had quantative exposure data of the workers to acrylonitrile and hence could not be utilized for calculation of a safe level. The criterion was therefore developed from the animal carcinogenicity data by utilizing the linear non-threshold model.

The rat carcinogenicity studies, in general, showed a tumorigenic response to acrylonitrile whether exposure was by ingestion or inhalation. These data support the findings of the epidemiological studies.

To select data for the evaluation of an acceptable risk concentration, studies having the following attributes were chosen:

1. There was an increase in frequency of tumors in treated rats over control rats.

2. There was a low frequency of tumors in control rats.

3. Several dosage levels were tested so dose-response relationships could be interpreted.

To use the linear dose-response non-threshold model in calculating a water concentration that results in a risk of a carcinogenicity incidence of 1/100,000, the following assumptions were made for all calculations:

1. A maximum bioaccumulation factor of 110 for acrylonitrile, as determined for the bluegill sunfish (EPA report, Duluth, Mn.).

2. Consumption of water per person per day is 2l over a period of 70 years.

3. Average consumption of fish per person per day is 18.7 grams.

4. Average life span for test rats is 730 days.

Specialized assumptions for converting inhalation dose to an equivalent ingestion dose were made with the Maltoni, et al. (1977) study as follows:

1. The average rat respiration rate is 0.61 1/min. kg (Guyton, 1947; Crosfill and Widdiconbe, 1961).

2. The average weight for male rats is 500 g and female rats 300 g.

3. The absorption efficiency is 90 percent (Young, et al. 1977).

4. Since the duration of the experiment was 1001 days, the average life span of the rat was assumed to be 1001 days due to constraints of the equations in the linear model.

5. The data of Young, et al. (1977) (see Pharmacokinetics) suggests that extrapolation from one dosage route to another may not be valid. Further verification of this data is needed, however, and the data of Maltoni's inhalation study was included for comparison with the water ingestion studies.

The results of the application of the linear non-threshold model to the selected data are summarized below:

| Reference | Route | Location of tumor | Sex | Dose (mg/kg) | Acceptable risk concentration in water (mg/l) |
|---|---|---|---|---|---|
| Norris, 1977 | Water ingestion | Proliferation lesions in brain. | Female | 12.45 | $1.2\times10^{-4}$ |
| | | Mammary gland | Female | 4.7 | $1.6\times10^{-4}$ |
| | | Ear canal masses | Female | 12.45 | $9.8\times10^{-4}$ |
| | | | Male | 23.8 | $8.8\times10^{-5}$ |
| Quast, et al | Water ingestion | Stomach | Female | 27.45 | $1.5\times10^{-4}$ |
| | | | Male | 23.8 | $8.8\times10^{-5}$ |
| | | Central nervous system. | Female | 11.4 | $8.3\times10^{-5}$ |
| | | | Male | 9.8 | $1.9\times10^{-4}$ |
| | | Zymbal gland | Female | 11.4 | $1.9\times10^{-4}$ |
| Maltoni, et al. 1977 | Inhalation | Gliomas | Male | *4.1 | $18.1\times10^{-4}$ |
| | | Mammary gland | Female | *1.0 | $0.6\times10^{-3}$ |
| | | | Male | *4.1 | $5.6\times10^{-4}$ |

*Calculated ingestion dose converted from inhalation.

In spite of the differences between the data sets, the application of linear non-threshold model results in relatively similar calculated acceptable risk concentrations for water in each case.

Due to the numerous assumptions necessary for the Maltoni study as well as the potential inability to convert between dosage routes, the data from this study were not weighed as heavily as the others. Therefore, the value of $0.8 \times 10^{-4}$ mg/l was selected as the recommended criterion for acrylonitrile in water. It must be emphasized that this level is based on data from a 12-month interim report and is, therefore, preliminary in nature.

Under the Consent Decree in NRDC vs. Train, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." Acrylonitrile is suspected of being a human carcinogen. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of acrylonitrile in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of acrylonitrile corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$ or $10^{-7}$ as shown in the table below.

| Exposure Assumptions | Risk Levels and Corresponding Criteria (1) | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ | $10^{-6}$ | $10^{-5}$ |
| 2 liters of drinking water and consumption of 18.7 grams of fish and shellfish (2) | | $0.008 \times 10^{-4}$ ng/l | $0.08 \times 10^{-4}$ ng/l | $0.8 \times 10^{-4}$ ng/l |
| Consumption of fish and shellfish only. | | $0.016 \times 10^{-4}$ ng/l | $0.16 \times 10^{-4}$ ng/l | $1.6 \times 10^{-4}$ ng/l |

(1) Calculated by applying a modified "one hit" extrapolation model described in the Methodology Document to the animal bioassay data presented in Appendix III. Since the extrapolation model is linear to low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1,000, and so forth.

(2) Fifty one percent of the acrylonitrile exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 110-fold. The remaining 49 percent of the acrylonitrile exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of acrylonitrile (1) occurring from the consumption of both drinking water and aquatic life grown in water containing the corresponding acrylonitrile concentrations and, (2) occurring solely from the consumption of aquatic life grown in the waters containing the corresponding acrylonitrile concentrations. Because data indicating other sources of exposure and the contribution to total body burden are inadequate for quantitative use, the criterion reflects the increment to risks associated with ambient water exposure only.

*Summary of Pertinent Data*

The water quality criterion for acrylonitrile is derived from the tumorigenic effect observed in the central nervous system of female Sprague-Dawley rats given 100 ppm acrylonitrile in drinking water. The time weighted average dose of 11.4 mg/kg/day was given for 52 weeks, and ten animals of each group were then sacrificed (interim). The incidence of brain tumors was 0/9 and 4/10 in the control and the treated groups, respectively. Assuming a fish bioconcentration of 110, the criterion is calculated from the following parameters:

$n_t = 4$
$N_t = 10$
$n_c = 0$
$N_c = 9$
$l_e = 368$ days
$L_e = 368$ days
$d = 11.4$ mg/kg/day
$R = 110$
$L = 730$ days
$w = 0.350$ kg
$F = 0.0187$ kg/day

Based on these parameters, the one-hit slope $B_H$ is 2.0455 $(mg/kg/day)^{-1}$. The resulting water concentration of acrylonitrile calculated to keep the individual risk below $10^{-5}$ is 0.084 µg/l. It must be emphasized that this concentration level is based on data from a 12-month interim report and is, therefore, likely to be modified when the final report becomes available.

**Aldrin/Dieldrin**

*Criteria Summary*

*Freshwater Aquatic Life.* For aldrin/dieldrin the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.0019 µg/l as a 24-hour average and the concentration should not exceed 1.2 µg/l at any time.

*Saltwater Aquatic Life.* For aldrin/dieldrin the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 0.0069 µg/l as a 24-hour average and the concentration should not exceed 0.16 µg/l at any time.

*Human Health.* For the maximum protection of human health from the potential carcinogenic effects of exposure to aldrin through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of aldrin estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria of $4.6 \times 10^{-2}$ ng/l, $4.6 \times 10^{-3}$ ng/l, and $4.6 \times 10^{-4}$ ng/l, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to dieldrin through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of dieldrin estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$, with corresponding criteria of $4.4 \times 10^{-2}$ ng/l, $4.4 \times 10^{-3}$ ng/l, and $4.4 \times 10^{-4}$ ng/l, respectively.

*Basis for the Criteria*

*Freshwater Aquatic Life. 1The maximum concentration of dieldrin is the Final Acute Value of 1.2* µg/l which is based on the more acutely sensitive invertebrate organisms. Since 0.44 times the Final Acute Value (0.44 x 1.2 µg/l=0.53 µg/l) is not lower than the Final Chronic Value (0.0019 µ1g/1), the latter is the recommended 24-hour average concentration. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

*Summary of Available Data.*

The concentrations below have been rounded to two significant figures.

Final Fish Acute value=1.6µg/l
Final Invertebrate Acute Value=1.2 µg/l
Final Acute Value=1.2µg/l
Final Fish Chronic Value=0.031µg/l
Final Invertebrate Chronic Value= not available
Final Plant Value=100 µg/l
Residue Limited Toxicant Concentration=0.0019 µg/l
Final Chronic Value=0.0019µg/l
0.44 × Final Acute Value=0.53µg/l

*Saltwater Aquatic Life.* No saltwater criterion can be derived for dieldrin using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available.

However, results obtained with dieldrin and freshwater organisms indicate how a criterion may be estimated. For freshwater organisms the Final Fish Chronic Value divided by the Final Fish Acute Value is 0.031/1.6=0.019. When this value is multiplied times the saltwater Final Fish Acute Value, an estimated Final Fish Chronic Value of 0.85×0.019=0.016 µg/l is obtained. Therefore, the Final Chronic Value of 0.0069 µg/l, based on the RLTC, should not cause adverse chronic effects on fish or invertebrate species.

To estimate a criterion for dieldrin, the maximum concentration is the Final Acute Value of 0.16 µg/l and the 24-hour average concentration is the Final Chronic Value of 0.0069 µg/l. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

*Summary of Available Data.* The concentrations below have been rounded to two significant figures.

Final Fish Acute Value=0.85 µg/l
Final Invertebrate Acute Value=0.16 µg/l
Final Acute Value=0.16 µg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=950 µg/l
Residue Limited Toxicant Concentration=0.0069 µg/l
Final Chronic Value=0.0069 µg/l
0.44×Final Acute Value=0.070 µg/l

*Human Health.* The aldrin and dieldrin carcinogenicity data of Walker, et al. (1972) and the National Cancer Institute (1976) were analyzed using a linear dose-response model to calculate that concentration of dieldrin in water which is estimated to result in an excess lifetime risk of $10^{-5}$ in man.

It should be noted that Walker, et al. study used 99 percent pure dieldrin while the NCI study used technical grade dieldrin.

Under the Consent Decree in NRDC vs. Train, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." Both aldrin and dieldrin are suspected of being human carcinogens. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of aldrin/dieldrin in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of aldrin and dieldrin corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$ or $10^{-7}$ as shown in the table below.

| Exposure Assumptions | Risk Levels and Corresponding Criteria (1) | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ | $10^{-6}$ | $10^{-5}$ |
| 2 liters of drinking water and consumption of 18.7 grams of fish and shellfish (2) | | | | |
| Aldrin | 0 | $4.6 \times 10^{-4}$ ng/l | $4.6 \times 10^{-3}$ ng/l | $4.6 \times 10^{-2}$ ng/l |
| Dieldrin | 0 | $4.4 \times 10^{-4}$ ng/l | $4.4 \times 10^{-3}$ ng/l | $4.4 \times 10^{-2}$ ng/l |
| Consumption of fish and shellfish only. | | | | |
| Aldrin | 0 | $4.6 \times 10^{-4}$ ng/l | $4.6 \times 10^{-3}$ ng/l | $4.6 \times 10^{-2}$ ng/l |
| Dieldrin | 0 | $4.5 \times 10^{-4}$ ng/l | $4.5 \times 10^{-3}$ ng/l | $4.5 \times 10^{-2}$ ng/l |

(1) Calculated by applying a modified "one hit" extrapolation model described in the 44 FR 15926, 1979. Appropriate bioassay data used in the calculation of the model are presented in the summary of pertinent data. Since the extrapolation model is linear to low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1,000, and so forth.

(2) 99.9 percent of aldrin exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 4500 fold. The remaining 0.1 percent of aldrin exposure results from drinking water.

Ninety-eight percent of dieldrin exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 4500 fold. The remaining 2 percent of dieldrin exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of aldrin/dieldrin, (1) occurring from the consumption of both drinking water and aquatic life grown in water

containing the corresponding aldrin/dieldrin concentrations and, (2) occurring solely from the consumption of aquatic life grown in the waters containing the corresponding aldrin/dieldrin concentrations.

Although total exposure information for aldrin and dieldrin is discussed and an estimate of the contributions from other sources of exposure can be made, this data will not be factored into the ambient water quality criteria formulation because of the tenuous estimates. The criteria presented, therefore, assume an incremental risk from ambient water exposure only.

*Summary of Pertinent Data for Aldrin.* The water quality criterion for aldrin is derived from the hepatocellular carcinoma response of the B6C3F1 male mice given the low dose of aldrin in the NCI bioassay test, and on the response in the 0.1 ppm group of female CF–1 mice in the Walker, et al. (1972) experiment. In the NCI study, a time-weighted average dose of 4 ppm was given in the feed for 80 weeks and the animals were observed for an additional 10 weeks before terminal sacrifice. The incidence of hepatocellular carcinoma was $^{3}/_{20}$ and $^{16}/_{49}$ in the control and treated groups, respectively. The slope of the one-hit dose-response curve for aldrin is calculated from the following parameters:

$n_t = 16$
$N_t = 49$
$n_c = 23$
$N_c = 20$
Le = 90 weeks
le = 80 weeks
d = 4 ppm × 0.13 = 0.52 mg/kg/day
L = 90 weeks
w = 0.035 kg

With these parameters the slope of the one-hit dose-response curve for aldrin is 6.349 $(mg/kg/day)^{-1}$.

The conversion of aldrin to dieldrin in fish results in the accumulation of dieldrin residues in fish exposed to aldrin. This makes it necessary to consider the risk resulting from intake of dieldrin stored in fish due to the presence of aldrin in water. Thus, the criterion for aldrin also depends upon the one-hit dose-response curve for dieldrin, which has a slope of 183.6 $(mg/kg/day)^{-1}$ as calculated previously from the Walker, et al. (1972) study.

The equation describing the risk due to aldrin in water is derived from the general relationship

$P = B_H D$ and $D = I/70$ kg, thus
$P = B_H\ I/70$ kg and
$P(70\ kg) = B_H I$

Where:

P = individual lifetime risk (set at $10^{-5}$ for criterion calculation)
I = average daily human intake of the substance in question
$B_H$ = average weight of humans

Since aldrin in water leads to the accumulation of dieldrin residues in fish, the equation describing the risk due to aldrin is

$Pa\ (70\ kg) = B_{Ha}\ C_a\ (2.0\ l/day) + B_{Ha}\ C_a\ R_{ad}\ (0.0187\ kg/day) + B_{Hd}\ C_a\ R_{ad}\ (0.0187\ kg/day)$

Where:

$P_a$ = risk due to aldrin (set at $10^{-5}$ for criterion calculation)
$B_{Ha}$ = 6.349 $(mg/kg/day)^{-1}$, the aldrin dose-response slope
$B_{Hd}$ = 183.6 $(mg/kg/day)^{-1}$, the dieldrin dose-response slope
$C_a$ = criterion concentration for aldrin (to be calculated)
$R_a$ = 32 l/kg, the fish bioconcentration of aldrin from aldrin
$R_{ad}$ = 4468 l/kg, the fish bioconcentration of dieldrin from aldrin
2.0 l/day = average daily intake of water for humans
0.0187 kg/day = average daily intake of fish for humans.

The term containing $R_{ad}$ represents intake of dieldrin resulting from the presence of aldrin in the water, and is thus multiplied by the dieldrin dose-response slope. $R_{ad}$ is estimated by assuming that in the absence of conversion to dieldrin, aldrin would bioconcentrate 4500 times (as dieldrin does), and that since aldrin only accumulates 32 times, the remainder of the expected aldrin residues are being stored as dieldrin.

The result is that the water concentration of aldrin should be less than $4.6 \times 10^{-2}$ ng/l in order to keep the individual lifetime risk below $10^{5}$.

*Summary of Pertinent Data for Dieldrin.* The water quality criterion for dieldrin is based on the hepatocellular carcinoma response of the female CF–1 mice given 0.1 ppm of dieldrin continuously in the diet in the experiment of Walker, et al. (1972). In that group the incidence of type a and type b liver tumors in the 0.1 ppm group of females was 24 out of 90 animals, whereas in the controls it was 39 out of 297 animals. Assuming a fish bioconcentration factor of 4500, the parameters of the dose-response model are:

$n_t = 24$
$N_t = 90$
$n_c = 39$
$N_c = 297$
Le = 132 weeks
le = 132 weeks
d = 0.1 ppm × 0.13 = 0.013 mg/kg/day
L = 132 weeks
w = 0.025 kg
R = 4500
F = 0.0187 kg/day

With these parameters the slope of the one-hit dose-response curve for dieldrin is 183.6 $(mg/kg/day)^{-1}$.

The result is that the water concentration should be less than $4.4 \times 10^{-2}$ ng/l in order to keep the individual lifetime risk below $10^{-5}$.

**Asbestos**

*Criteria Summary*

*Freshwater Aquatic Life.* For freshwater aquatic life, no criterion for asbestos can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Saltwater Aquatic Life.* For saltwater aquatic life, no criterion for asbestos can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* For the maximum protection of human health from the potential carcinogenic effects of exposure to asbestos through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of asbestos estimated to result in additional lifetime cancer risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, $10^{-7}$ with corresponding criteria of 300,00 fibers/l, 30,000 fibers/l, and 3,000 fibers/l, respectively.

*Basis for the Criteria*

*Aquatic Life.* No appropriate data on the effects of asbestos on aquatic organisms are available at this time. Therefore, no freshwater or saltwater criterion can be derived for asbestos using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* A substantial body of data exists which shows increased incidence of cancer of the esophagus, stomach, colon, and rectum or peritoneal mesothelioma in humans exposed to asbestos occupationally. For several of these groups, data exist on the approximate air-borne fiber concentrations to which individuals were exposed. These human data will serve as the primary basis for a standard of asbestos in water. Experimental data indicate that a major fraction of the asbestos deposited in the lungs is subsequently swallowed. In this section the dose to the gastrointestinal tract of four occupational groups will be calculated from knowledge of the air

concentrations to which the workers were exposed and follow the assumption that all the asbestos inhaled subsequently passed through the gastrointestinal tract and provided the exposure that led to the observed increase in abdominal cancer. The assumption that all inhaled asbestos ingested is an overestimate but not significantly. No account has been taken of the material that a worker may swallow directly, and this quantity could be significant. However, for the purposes of a criterion, our inability to quantiate direct ingestion in the work place and to properly account for it by a the present approach provides some margin of safety in the estimate of dose-response relations.

Table 1 lists the percentage of death from excess gastrointestinal cancer and peritoneal mesothelioma in four groups of asbestos workers. Calculations of this percentage were made using expected numbers of death, rather than the observed, because the latter was inflated by including other asbestos-related deaths.

**Table I.**—*Percentage of Excess Gastrointestinal Cancers and Peritoneal Mesothelionias in Four Groups of Asbestos Workers*

| Exposed group | Number of excess deaths (from table 28) | | Expected number of deaths in cohort | Excess deaths as a percentage of expected deaths in cohort | | |
|---|---|---|---|---|---|---|
| | G.I. cancer | Peritoneal mesothelioma | | G.I. | Peritoneal mesothelioma | Total |
| Insulation workers [a] (chrysotile and amosite) | 39.9 (ICD 150–154) [e] | 109 | 1660.96 | 2.4 | 6.6 | 9.0 |
| Insulation workers [b] (chrysotile and amosite) | 29.4 (ICD 150–154) | 22 | 305.20 | 9.6 | 7.2 | 16.8 |
| Factory employment [c] (amosite) | 10.5 (ICD 150–154) | 8 | 368.62 | 2.9 | 2.2 | 5.1 |
| Factory employment [d] (chrysotile, crocidolite and amosite) | 15.8 (ICD 150–154 ex meso) | 35 | 556.0 | 2.8 | 6.3 | 9.1 |

[a] Selikoff, et al. (1979).
[b] Selikoff, et al. (1976).
[c] Seidman, et al. (1979).
[d] Newhouse and Berry (1979).
[e] Public Health Service (1967–69).

Table 2 lists the fiber concentration estimates and an exposure index for each cohort (years of exposure *x* fiber concentration). This index will be used to calculate the number and mass of asbestos fibers ingested during a working lifetime. As the observed mortality is, to a large extent, after 20 years from first exposure, the intermixing of time and exposure does not present significant problems.

The average length of exposure for the insulation workers in the first group was calculated from data on employment time at entry into the cohort in 1967. Forty years was used as the working lifetime for the smaller group of New York and New Jersey insulators, virtually all of whom are deceased or retired. The estimate of the person-weighted exposure index for the amosite factory is simply the average employment time multiplied by 40 f/ml. Data from Table 3 were used to estimate a person-weighted exposure index for the Newhouse and Berry group.

**TABLE 2.**—*Exposure Indices for Asbestos Worker Groups*

| Exposed group | Air fiber concentration (f/ml) | Person-weighted exposure average time (yrs.) | Exposure index (years × f/ml) |
|---|---|---|---|
| U.S. Insulators Selikoff, et al. (1979) | 15 | 34 | 510 |
| NY/NJ insulators Selikoff, et al. (1976) | 15 | 40 | 600 |
| Amosite factory workers Seidman, et al. (1979) | 40 | 1.9 | 76 |
| British factory workers Newhouse and Berry (1979) | 10–30 | (1) | 180 |

[1] See table 3c

(Person-weighted exposure index = $\frac{\text{(No. at risk} \times \text{exposure} \times \text{time)}}{\text{(No. at risk)}}$ = 180.)

**Table 3.**—*Exposure Estimates for Workers in a British Factory* [a]

| Exposure group | No. at risk | Exposure (f/ml) | Time of exposure (years) |
|---|---|---|---|
| Severe: | | | |
| 2 years | 711 | 30 | 20 |
| 2 years | 1,333 | 30 | 2 |
| Low to moderate: | | | |
| 2 years | 503 | 10 | 20 |
| 2 years | 933 | 10 | 2 |

[a] Newhouse and Berry (1979).

A detailed calculation of the daily intake of asbestos to produce a lifetime risk of $10^{-5}$ is given in Appendix I. Data of the occupational risk of both gastrointestinal cancer and peritoneal mesothelioma were used (Table 1). Account was taken of the fact that occupational exposures took place over a 5-day work week and that the ingestion exposure may encompass a lifespan of 70 years. It was assumed that a worker breathes at the rate of 1 $m^3$/hr during work exposure for the purpose of calculating total asbestos intake per day. Using a linear dose-response relationship, and a specified risk of $10^{-5}$, the calculated 70-year daily intake resulting from these calculations are given in Table 4. The data from Seidman, et al. were not used because it was exclusively from amosite exposures. Assuming that two liters of water are ingested per day, this would correspond to a concentration of 300,000 fibers of all sizes/liter of water.

**Table 4.**—*Calculated Intake for $10^{-5}$ Lifetime Risk of Death From Gastrointestinal Cancer and Peritoneal Mesothelioma*

| Exposure group | Estimate of intake/day for $10^{-5}$ risk (fibers of all lengths/day) |
|---|---|
| Selikoff, et al. (1979) | 900,000 |
| Selikoff, et al. (1976) | 600,000 |
| Newhouse and Berry, (1979) | 400,000 |
| Average | 600,000 |

A criterion for a mass concentration of asbestos can also be calculated using the conversion value of 30 $\mu g/m^3/f/ml$ as derived from the data for predominantly chrysotile exposures. A value of 150 $\mu g/m^3/f/ml$ for amosite appears more appropriate, based on the finding that amosite has approximately a three time greater conversion factor than chrysotile. A detailed calculation is given in the criteria document and the results are summarized in Table 5. Assuming that two liters of water are ingested per day, a risk of $10^{-5}$ would be produced from ingesting water

containing 0.05μg/liter. As mentioned in the criteria document, the variability in the data used to convert optical fiber counts to mass leads to a large uncertainty in the above estimate.

**Table 5.**—*Calculated Intake for $10^{-4}$ Lifetime Risk of Death From Gastrointestinal Cancer and Peritoneal Mesothelioma*

| Exposure group | Estimate of intake/day for $10^{-5}$ risk (μg/l day) |
|---|---|
| Selikoff, et al. (1979) | 0.14 |
| Selikoff, et al. (1976) | 0.09 |
| Seidman, et al. (1979) | 0.11 |
| Newhouse and Berry, (1979) | 0.05 |
| Average | 0.1 |

Considering chrysotile and depending on the source of the abestos in water, 0.05 μg/l corresponds to from $10^6$ to $20 \times 10^6$ fibers of all lengths per day. Such estimates are considerably higher than those derived previously and are most likely a reflection of the differences in the sizes of the fibers found in water, as compared to those found in air. Because of these uncertainties, high priority should be given to obtaining accurate size and mass distribution of typical fibers found in different circumstances (air and water) which would allow appropriate conversions to be made between fiber concentrations in air and water.

The majority of samples analyzed for the EPA to date were characterized by a concentration of all microscopic visible fibers per liter of water.

Further, techniques for the determination of fiber concentrations (as opposed to mass concentrations) have been published as interim EPA procedures. Thus, a criterion for the concentration of fibers of all sizes in water corresponding to a $10^{-5}$ risk will be calculated directly from the concentrations of fibers greater than 5 μm measured in the occupational circumstances that produced disease. Unfortunately, the data currently available relating concentrations of fibers longer than 5μm, counted by optical microscopy, determined by electron microscopy, are extremely limited. These include those by Wallingford (1978), 15:1, Millette (personal communication), 400:1; and Winer and Cossett (1978), 1000:1 and are only for chrysotile asbestos. Using the geometric mean of 200 for this factor from all available data, a total fiber concentration corresponding to a $10^{-5}$ risk can be calculated from the data of Tables 1 and 2.

In making the calculation, one tacitly assumes the same fiber size distribution in water as in occupational air samples. Some data show that water fiber size distributions vary, and occupational air distributions have been shown to be so variable that the fraction of fibers longer than 5 microns can range over a factor of 10 depending on sampling circumstances. Although sizing of airborne and waterborne fibers have not been done using the same methods, qualitatively, water appears to have fiber distributions with more smaller fibers than in occupational air samples. Thus, an estimate assuming the same fiber size distribution in water as in air will yield a conservate criterion (from the point of view of health).

Although positive animal experiments had various experimental limitations, such data as existed were treated in the model of EPA. The data are presented in Table 6.

**Table 6**[2]

| Effect | Estimated $10^{-5}$ dosage(μg/l) |
|---|---|
| 4/42 Kidney carcinomas 0/49 control | 3.2 |
| 12/42 Malignancies 2/49 control | 1.1 |

[2] Gibel, et al. (1976).

Considering the large number of experimental uncertainties, these values provide reasonable support for the concentration derived from human exposure data.

This document was concerned with the estimation of that concentration of asbestos in water which will produce a lifetime risk of 1 in 100,000 in a population exposed continuously. The risk estimate was made using a linear extrapolation from existing human data and would appear to constitute a conservative extrapolation. However, in the case of asbestos the risk factor of 1/100,000 is not conservative. If we were concerned with intermittent or localized contamination incidents of some carcinogen that, once identified, could be abated, such a value would have utility. With asbestos, however, we are concerned with an ubiquitous contaminant in the environment to which large populations are continuously exposed for decades. Further, the estimated value has a high degree of uncertainty associated with it, based upon the data from which it was derived.

Under the Consent Decree in *NRDC* v. *Train*, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." Asbestos is suspected of being a human carcinogen. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of asbestos in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of asbestos corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$, or $10^{-7}$ as shown in the table below.

| Exposure Assumption | Risk Levels and Corresponding Criteria | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ | $10^{-6}$ | $10^{-5}$ |
| 2 liters of drinking water | | 3,000 f/1* | 30,000 f/1 | 300,000 f/1 |
| Consumption of fish and shellfish only. | | No Criterion | | |

*f = fibers

(1) Calculated by applying a modified "one-hit" extrapolation model described in the Methodology Document to the human epidemiological data presented in criterion document. Since the extrapolation model is linear to low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations

shown in the table by factors such as 10, 100, 1,000, and so forth.

Concentration levels were derived assuming a lifetime exposure to various amounts of asbestos occurring from the consumption of drinking water only.

Although total exposure information for asbestos is discussed and an estimate of the contributions from other sources of exposure can be made, this data will not be factored into ambient water quality criteria formulation until additional analysis can be made. The criteria presented, therefore, assume an incremental risk from ambient water exposure only.

**Benzidine**

*Criteria Summary*

*Freshwater Aquatic Life.* For freshwater aquatic life, no criterion for benzidine can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Saltwater Aquatic Life.* For saltwater aquatic life, no criterion for benzidine can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* For the maximum protection of human health from the potential carcinogenic effects of exposure to benzidine through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of benzidine estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion Formulation section of this document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$, with corresponding criteria of $1.67\times10^{-3}$ µg/l, $1.67\times10^{-4}$ µg/l, and $1.67\times10^{-5}$ µg/l, respectively.

*Basis for the Criteria*

*Freshwater and Saltwater Aquatic Life.* No freshwater or saltwater criterion can be derived for benzidine using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* The available data concerning the carcinogenicity of benzidine in experimental animals are severely limited. It is extremely difficult to extrapolate the experimental results to man because, with the possible exception of the dog and the rabitt, the target organs are different. Moreover, the metabolites produced by the various species, in general, differ significantly from those produced by man (Haley, 1975), although 3-hydroxybenzidine and its conjugation products are common to both man and animals.

Despite the limitations of the available data, a suggested criterion for benzidine was calculated using the linear non-threshold model described in 44 FR 15926 March 15, 1979. The calculation assumes a risk of 1 in 100,000 of developing cancer as a result of daily consumption of 2 liters of benzidine contaminated water and the daily consumption of 18.7 g of benzidine contaminated aquatic organisms. Based on the data of Zavon, et al. (1973)[1], a benzidine criterion of $1.67\times10^{-3}$, µg/l is suggested to be adequate to protect the population consuming the water and the contaminated aquatic organisms.

Epidemiological data indicate that exposure to benzidine is associated with an increase in bladder cancer in man. The possibility that benzidine may be found in wastewater may also pose a problem. In order to determine the extent of the potential problem, measurements must be made of wastewater not only for benzidine but also for its congeners. Moreover, further evaluation must be made on these chemicals and their azo dye derivatives to determine their stability to microbiological degradation. It is essential that studies of their carcinogenicity in experimental animals be made at doses which produce a bare minimum of liver pathology. A detailed pharmacokinetic study should be undertaken to establish routes of absorption, body transport, storage and excretion of benzidine, its congeners, and the azo dyes synthesized from them. Programs covering both industrial hygienic and epidemiologic aspects of exposure to benzidine and its congeners to establish the degree of dermal and pulmonary absorption are a necessity if we are to prevent this chemically induced cancer from occurring.

Under the Consent Decree in *NRDC vs. Train*, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." Benzidine is suspected of being a human carcinogen. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of benzidine in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of benzidine corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$ or $10^{-7}$ as shown in the table below.

| Exposure Assumptions | Risk Levels and Corresponding Criteria (1) | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ | $10^{-6}$ | $10^{-5}$ |
| 2 liters of drinking water and consumption of 18.7 grams of fish and shellfish (2) | 0 | $1.67 \times 10^{-5}$ µg/l | $1.67 \times 10^{-4}$ µg/l | $1.67 \times 10^{-3}$ µg/l |
| Consumption of fish and shellfish only. | 0 | $5.24 \times 10^{-5}$ µg/l | $5.24 \times 10^{-4}$ µg/l | $5.24 \times 10^{-3}$ µg/l |

(1) Calculated by applying a modified "one hit" extrapolation model described in 44 FR 15926 March 15, 1979. Appropriate epidemiological data used in the calculation of the model are presented in Summary of Pertinent Data. Since the extrapolation model is linear to low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other

[1] Zavon, M.R., et al. 1973. Benzidine exposure as a cause of bladder tumors. Arch. Environ. Health 27:1.

risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1,000, and so forth.

(2) Thirty-two percent of benzidine exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 50-fold. The remaining 68 percent of benzidine exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of benzidine, (1) occurring from the consumption of both drinking water and aquatic life grown in water containing the corresponding benzidine concentrations and, (2) occurring solely from the consumption of aquatic life grown in the waters containing the corresponding benzidine concentrations.

Although total exposure information for benzidine is discussed and an estimate of the contributions from other sources of exposure can be made, this data will not be factored into the ambient water quality criteria formulation because of the tenuous estimates. The criteria presented, therefore, assume an incremental risk from ambient water exposure only.

*Summary of Pertinent Data*

The data from the human epidemiology study of Zavon, et al. 1973 [1] were used to estimate the concentration of benzidine in water calculated to keep the lifetime cancer risk below $10^{-5}$. In this study 25 workers in a benzidine manufacturing plant were observed for the appearance of bladder tumors after a mean exposure period of 13.61 years, their average age at the end of exposure was 44 years and at the end of a 13-year observation was 57 years. The men not showing evidence of cancer had a mean exposure period of 8.91 years, their average age at the end of exposure was 43 years and at the end of observation 56 years. The estimated total accumulated dose of 200 mg/kg was estimated from average urinary levels of benzidine in these workers at the end of a workshift (see Zavon, et al. 1973).[1] The criterion was calculated from the following parameters:

Average weight of man = 70 kg
Observed incidence of bladder cancer = 13/25 (52 percent)
Accumulated dose = 200 mg/kg
Bioconcentration factor of benzidine = 50
X = average daily exposure producing lifetime risk of $10^{-5}$
$B^*$ = potency factor, which is an estimate of the linear dependency of cancer rates on lifetime average dose
C = concentration of benzidine in water, calculated to produce a lifetime risk of $10^{-5}$, assuming a daily ingestion of 2 liters of water and 0.0187 kg fish.

Workers were assumed to have received 200 mg/kg of benzidine in a lifetime. At the end of a 13-year observation period, the average age of the workers was 57 years. Therefore, benzidine exposure on a mg/day basis amounts to:

$$\frac{200 \times 70}{365 \times 57} = .673 \text{ mg/day}$$

This gives a response at 57 years of 52% so that:

$$.52 = 1 - e^{-B(.673)}$$
$$B = \frac{.734}{.673} = 1.091$$

$$B^* = B\left(\frac{tf}{tf}\right)^3 = 1.091\left(\frac{70}{57}\right)^3 = 2.021$$

$$(2.021)\ (X) = 10^{-5}$$
$$X = 4.9 \times 10^{-6} \text{ mg/day to obtain a rate of } 10^{-5} \text{ or } 4.9 \times 10^{-3} \ \mu g/day$$

Therefore:

$$C(2 + 50 \times .0187) = 4.9 \times 10^{-3}$$
$$C = 1.67 \times 10^{-3} \ \mu g/l$$

From this data the concentration of benzidine in water calculated to keep lifetime cancer risk below $10^{-5}$ is $1.67 \times 10^{-3}$ µg/l.

**Chloroalkyl Ethers**

*Criteria Summary*

*Freshwater Aquatic Life.* For freshwater aquatic life, no criterion for any chloroalkyl ether can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Saltwater Aquatic Life.* For saltwater aquatic life, no criterion for any chloroalkyl ether can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* For the protection of human health from the toxic properties of bis(2-chloroisopropyl) ether ingested through water and through contaminated aquatic organisms, the ambient water criterion is determined to be 175.8 µg/l. For the maximum protection of human health from the potential carcinogenic effects of exposure to bis(2-chloroisopropyl) ether through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of bis(2-chloroisopropyl) ether estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$, with corresponding criteria of 11.5 µg/l, 1.15 µg/l, and 0.115 µg/l, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to bis(2-chloroethyl) ether through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of bis(2-chloroethyl) ether estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria of 0.42 µg/l, 0.042 µg/l, and 0.0042 µg/l, respectively.

For the maximum protection of human health from the potential carcinogenic effects of exposure to bis(chloromethyl) ether through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of bis(chloromethyl) ether estimated to result in additional lifetime cancer risks ranging from no

additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria of 0.02 ng/l, 0.002 ng/l, and 0.0002 ng/l, respectively.

*Basis for the Criteria*

*Freshwater Aquatic Life.* No freshwater criterion can be derived for any chloroalkyl ether using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

*Summary of Available Data.* The concentrations below have been rounded to two significant figures.

*2-chloroethyl vinyl ether*

Final Fish Acute Value = 50,000 μg/l
Final Invertebrate Acute Value = not available
Final Acute Value = 50,000 μg/l
Final Fish Chronic Value = not available
Final Invertebrate Chronic Value = not available
Final Plant Value = not available
Residue Limited Toxicant Concentration = not available
Final Chronic Value = not available
0.44 × Final Acute Value = 22,000 μg/l

*bis (2-chloroethyl) ether*

Final Fish Acute Value = not available
Final Invertebrate Acute Value = 9,600 μg/l
Final Acute Value = 9,600 μg/l
Final Fish Chronic Value = greater than 1,400 μg/l
Final Invertebrate Chronic Value = not available
Final Plant Value = not available
Residue Limited Toxicant Concentration = not available
Final Chronic Value = greater than 1,400 μg/l
0.44 × Final Acute Value = 4,200 μg/l

*Saltwater Aquatic Life.* No saltwater criterion can be derived for any chloroalkyl ether using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* There is no empirical evidence that BCIE is carcinogenic; however, some chronic toxic effects of the compound have been noted. One approach to estimating a safe level of BCIE in drinking water utilizes the following general equation:

$$NOAEL \times SF \times BW = W \times Z + R \times F \times Z + AD - (R \times F \times Z)$$

where NOAEL = no apparent adverse effect level in mammals
SF = safety factor
BW = body weight of average human (assume 70 kg)
W = daily consumption of water (assume 2 liters)
Z = safe level for water
R = bioconcentration factor (in l/kg)
F = daily consumption of fish (assume 0.0187 kg)
A = daily amount absorbed from air
D = daily amount from total diet (including fish)

Since valid estimates on current exposure from air and total diet cannot be made, the equation can be simplified to $NOAEL \times SF \times BW = (W + R \times F) \times Z$. The lowest dose tested which caused minimum adverse effects was 10 mg/kg/day for the mice. However, even at this dose, there was an increased incidence of centrilobular necrosis of the liver which was not seen in the high-dose group. To be conservative, a safety factor of $\frac{1}{1000}$ will be applied. Assuming an average human body weight of 70 kg, acceptable daily intake calculated is 700 μg/day. Using the estimated bioconcentration factor of 106 for BCIE and assuming daily consumption of 0.0187 kg fish and 2 liters of water, the safe level calculated from these data is 175.8 μg/l. Since this safe level is calculated on the basis of several assumptions that cannot be defended, it should be regarded as a very crude estimate.

Another approach to deriving a criterion has been suggested by the Carcinogens Assessment Group, EPA.

As previously states, BCIE has not been empirically proven to be a carcinogen; nevertheless, it is mutagenic and is in a class of compounds that are known as carcinogens. Based on these facts, credence can be lent to deriving a suggested criterion based upon NCI preliminary data (1978) as applied to the linear, non-threshold model.

Therefore, a lower bound water concentration of 11.5 μg/l has been calculated such that there is a 95 percent confidence that this level is lower than the actual level which would produce a $10^{-5}$ lifetime cancer risk due to exposure to BCIE.

Although both approaches to calculating a criterion are somewhat tenuous, the weight of evidence for the carcinogenic potential of BCIE is sufficient to be "qualitatively suggestive" and must not be ignored from a public health point of view. Until further conclusive data become available, the Agency feels it is prudent to consider BCIE as a potential carcinogen.

The estimated safe level of BCEE in drinking water may be caluculated using the same linear, non-threshold model as applied to BCIE. The data on the carcinogenicity of this compound by oral administration to male mice are used in the calculation. The bio-accumulation factor used is 25. Based on this approach, the calculated water quality criterion for BCEE is .42 μg/l. Compliance to this level should limit human lifetime risk of carcinogenesis from BCEE in drinking water to not more than $10^{-5}$ (one case in 100,000 persons at risk), assuming water to be the only source of exposure. It should also very adequately protect against noncarcinogenic toxicity since the daily dose of contaminant that would be absorbed from water containing the criterion limit is many times less than the minimal daily oral dose required to produce a detectable toxic response in animals.

The setting of drinking water standards for BCME and CMME is of academic interest only, since these α-chloroalkyl ethers may not, under ordinary conditions, exist in water for periods of time longer than a few hours. Carcinogenicity data generated by oral administration of these compounds are not available.

In the case of CMME, no criterion was calculated due to its extremely short half-life in aqueous solution. The hydrolysis rate of CMME in aqueous isopropanol has been measured. Extrapolation of the data to pure water yielded a $t_{1/2}$ of less than 1 second. BCME has a slightly longer half-life. Therefore, as a guideline, the safe level of BCME in drinking water may be calculated using the tumor incidence data from chronic rat inhalation studies (Kuschner, et al. 1975). In this study, Sprague-Dawley rats were exposed to 0.1 ppm BCME 6 hours per day, 5 days per week throughout their lifetime. Additional groups of rats were given 10, 20, 40, 80, and 100 exposures to 0.1 ppm BCME. The validity of the incidence rates for humans was established by evaluating the cancer incidence in workers after accounting for their exposure.

Therefore, using the linear, non-threshold model and a bioconcentration factor of 31, the recommended maximum permissible concentration of BCME for the ingested water is .02 ng/l. Compliance to this level should limit human lifetime risk of carcinogenesis from BCME in drinking water to not more than $10^{-5}$, assuming water to be the only source of exposure.

Under the Consent Decree in *NRDC vs. Train*, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities."

BCIE, BCEE, and BCME are suspected of being human carcinogens. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of these chloroalkyl ethers in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of BCIE, BCEE, and BCME corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$, or $10^{-7}$ as shown in the following table.

| Exposure assumptions | | Risk levels and corresponding criteria[1] | | |
|---|---|---|---|---|
| | | $10^{-7}$ (μg/l) | $10^{-6}$ (μg/l) | $10^{-5}$ (μg/l) |
| 2 liters of drinking water and consumption of 18.7 grams of fish and shellfish:[2] | | | | |
| Bis(2-chloroisopropyl)ether | [2] | 0.115 | 1.15 | 11.5 |
| Bis(2-chloroethyl)ether | 0 | 0.0042 | 0.042 | 0.42 |
| Bis(chloromethyl)ether | [2] | $0.02\times10^{-5}$ | $0.02\times10^{-4}$ | $0.02\times10^{-3}$ |
| Consumption of fish and shellfish only: | | | | |
| Bis(2-chloroisopropyl)ether | [2] | 0.231 | 2.31 | 23.1 |
| Bis(2-chloroethyl)ether | 0 | 0.0219 | 0.219 | 2.19 |
| Bis(chloromethyl)ether | 0 | $0.09\times10^{-5}$ | $0.09\times10^{-4}$ | $0.09\times10^{-3}$ |

[1] Calculated by applying a modified "one hit" extrapolation model described in the 44 FR 15926. Appropriate bioassay data used in the calculation of the model are presented in the summary of pertinent data. Since the extrapolation model is linear to low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1,000, and so forth.

[2] Fifty percent of BCIE exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 106-fold. The remaining 50 percent of BCIE exposure results from drinking water.

Nineteen percent of BCEE exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 25-fold. The remaining 81 percent of BCEE exposure results from drinking water.

Twenty-two percent of BCME exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 31-fold. The remaining 78 percent of BCME exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of BCIE, BCEE, and BCME, (1) occurring from the consumption of both drinking water and aquatic life grown in water containing the corresponding chloroalkyl ether concentrations and, (2) occurring solely from consumption of aquatic life grown in the waters containing the corresponding chloroalkyl ether concentrations.

Although total exposure information for these chloroalkyl ethers is discussed and an estimate of the contributions from other sources of exposure can be made, these data will not be factored into the ambient water quality criteria formulation because of the tenuous estimates. The criteria presented, therefore, assume an incremental risk from ambient water exposure only.

*Summary of Pertinent Data*

*Bis (2-Chloroisopropyl) Ether.* A 95 percent lower bound estimate of the water concentration of BCIE producing $10^{-5}$ cancer risk is calculated from the preliminary data of the NCI study in Osborne-Mendel rats. Since there is no statistically significant tumor incidence in any treated group compared with controls, the incidence of total malignant tumors in the male rats of the low dose group is compared with that of the respective vehicle control male group. The low dose group was given 100 mg/kg/day of BCIE by intubation 5 days per week for 2 years, so that the average lifetime exposure was 71.4 mg/kg/day. The lower bound water concentration is calculated from the values and the equation shown below. To obtain an upper 95 percent confidence bound on the slope, the following estimate was used

$$B_a u = \ln\left[\frac{1 - P_c(1)}{1 - P_t(u)}\right]$$

where $P_c(1)$ is the lower 2.5 percent confidence limit on the control malignant tumor rate and $P_t(u)$ is the upper 97.5 percent confidence bound on the malignant tumor rate in the treated group.

$n_t=17$ $l_e=104$ wk
$N_t=50$ $L=104$ wk
$n_c=22$ $d=71.4$ mg/kg/day
$N_c=50$ $w=.550$ kg
$L_e=104$ wk $F=.0187$ kg
$R=106$

Based on these parameters, the upper 95 percent confidence limit on the one-hit slope ($B_H u$) is $1.53\times10^{-2}$ (mg/kg/day)$^{-1}$. Therefore, the 95 percent lower bound estimate of the water concentration of BCIE producing $10^{-5}$ lifetime cancer risk is 11.5 micrograms per liter.

*Bis (2-Chloroethyl) ether.* The water quality criterion for BCEE is based on the induction of hepatomas in male mice (strain C57BL/6×C3H/Anf)$F_1$) given a daily oral dose of 300 ppm for 80 weeks (Innes, et al. 1969). The tumor incidence was 14/16 in the treated group compared with 8/79 in the control group. The criterion was calculated from the following parameters.

$n_t=14$ $L=80$ wk
$N_t=16$ $d=300$ ppm$\times0.13=39$ mg/kg/day
$n_c=8$ $w=.030$ kg
$N_c=79$ $F=.0187$ kg
$L_e=80$ wk $R=25$
$l_e=80$ wk

Based on these parameters, the one-hit slope ($B_H$) is $6.8510\times10^{\times1}$ (mg/kg/day)$^{-1}$. The resulting water concentration of BCEE calculated to keep the individual lifetime cancer risk below $10^{-5}$ is 0.42 micrograms per liter.

*Bis (Chloromethyl) Ether.* The water quality criterion for BCME is based on the inducation of malignant respiratory tract tumors in male Sprague-Dawley rats given 100 exposures of 0.1 ppm by inhalation 6 hours per day, 5 days per week (Kuschner, et al. 1975). The average lifetime exposure was calculated to be $3.510\times10^{-4}$ mg/kg/day. The tumor incidence was 12/20 in the treated group and 0/240 in the control rats. The criterion was calculated from the following parameters.

$n_t=12$
$N_t=20$
$n_c=0$
$N_c=240$
$L_e=104$ wk
$l_e=104$ wk
$L=104$ wk
$d=3.510\times10^{-4}$ mg/kg/day
$w=.500$ kg
$F=.0187$ kg
$R=31$

Based on these parameters, the one-hit slope ($B_H$) is $1.3603\times10^{4}$ (mg/kg/day)$^{-1}$. The resulting water concentration of BCME calculated to maintain the individual lifetime cancer risk below $10^{-5}$ is 0.02 nanograms per liter.

**Chlorinated Benzenes**

*Criteria Summary*

Freshwater Aquatic Life

*Chlorobenzene.* For chlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 1,500 μg/l as a 24-hour average and the concentration should not exceed 3,500 μg/l at any time.

*1,2,4-trichlorobenzene.* For 1,2,4-trichlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 210 μg/l as a 24-hour average and the concentration should not exceed 470 μg/l at any time.

*1,2,3,5-tetrachlorobenzene.* For 1,2,3,5-tetrachlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 170 μg/l as a 24-hour average and the concentration should not exceed 390 μg/l at any time.

*1,2,4,5-tetrachlorobenzene.* For 1,2,4,5-tetrachlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 97 μg/l as a 24-hour average and the concentration should not exceed 220 μg/l at any time.

*Pentachlorobenzene.* For pentachlorobenzene the criterion to protect fresh-water aquatic life as derived using procedures other than the Guidelines is 16 μg/l as a 24-hour average and the concentration should not exceed 36 μg/l at any time.

Saltwater Aquatic Life

*Chlorobenzene.* For chlorobenzene the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 120 μg/l as a 24-hour average and the concentration should not exceed 280 μg/l at any time.

*1,2,4-trichlorobenzene.* For 1,2,4-trichlorobenzene the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 3.4 μg/l as a 24-hour average and the concentration should not exceed 7.8 μg/l at any time.

*1,2,3,5-tetrachlorobenzene.* For 1,2,3,5-tetrachlorobenzene the criterion to protest saltwater aquatic life as derived using procedures other than the Guidelines is 2.6 μg/l as a 24-hour average and the concentration should not exceed 5.9 μg/l at any time.

*1,2,4,5-tetrachlorobenzene.* For 1,2,4,5-tetrachlorobenzene the criterion to protect saltwater aquatic life as derived using the Guidelines is 9.6 μg/l as a 24-hour average and the concentration should not exceed 26 μg/l at any time.

*Pentachlorobenzene.* For pentachlorobenzene the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 1.3 μg/l as a 24-hour average and the concentration should not exceed 2.9 μg/l at any time.

Human Health

For the prevention of adverse organoleptic or toxicological effects, the recommended criteria for chlorinated benzenes are as follows:

| Substance | Criterion μg/l | Basis for criterion |
|---|---|---|
| Monochlorobenzene [1] | 20 | Organoleptic effects. |
| Trichlorobenzene | 13 | Organoleptic effects. |
| Tetrachlorobenzene | 17 | Toxicity studies. |
| Pentachlorobenzene | .5 | Toxicity study. |

[1] A toxicological evaluation of monochlorobenzene resulted in a level of 450 μg/l; however, organoleptic effects have been reported at 20 μg/l.

For the maximum protection of human health from the potential carcinogenic effects of exposure to hexachlorobenzene (HCB) through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of HCB estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria of 1.25 ng/l, 0.125 ng/l, and 0.0125 ng/l, respectively.

*Basis for the Criteria*

*Freshwater Aquatic Life.* No freshwater criterion can be derived for any chlorinated benzene using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

However, data for 1,2,4,5-tetrachlorobenzene and saltwater organisms and 1,2-dichlorobenzene and freshwater organisms can be used as the basis for estimating criteria.

For 1,2,4,5-tetrachlorobenzene and saltwater organisms 0.44 times the Final Acute Value is 11 μg/l. This concentration is closed to the Final Chronic Value of 9.6 μg/l derived from an embryo-larval test with the sheepshead minnow. Also, for 1,2-dichlorobenzene and freshwater organisms 0.44 times the Final Acute Value is less than the Final Chronic Value based on an embryo-larval test with the fathead minnow. Therefore, a reasonable estimate of criteria for chlorinated benzenes and freshwater organisms would be 0.44 times the Final Acute Value.

*Chlorobenzene.* The maximum concentration of chlorobenzene is the Final Acute Value of 3,500 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For chlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 1,500 μg/l as a 24-hour average and the concentration should not exceed 3,500 μg/l at any time.

*1,2,4-trichlorobenzene.* The maximum concentration of 1,2,4-trichlorobenzene is the Final Acute Value of 470 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,2,4-trichlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other then the Guidelines is 210 μg/l as a 24-hour average and the concentration should not exceed 470 μg/l at any time.

*1,2,3,5-tetrachlorobenzene.* The maximum concentration of 1,2,3,5-tetrachlorobenzene is the Final Acute Value of 390 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,2,3,5-tetrachlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 170 μg/l as a 24-hour average and the concentration should not exceed 390 μg/l at any time.

*1,2,4,5-tetrachlorobenzene.* The maximum concentration of 1,2,4,5-tetrachlorobenzene is the Final Acute Value of 220 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,2,4,5-tetrachlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 97 μg/l as a 24-hour average and the concentration should not exceed 220 μg/l at any time.

*Pentachlorobenzene.* The maximum concentration of pentachlorobenzene is the Final Acute Value of 36 μg/l and the estimated 24-hour average concentration

is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For pentachlorobenzene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 16 μg/l as a 24-hour average and the concentration should not exceed 36 μg/l at any time.

*Summary of Available Data*

The concentrations below have been rounded to two significant figures.

*Chlorobenzene*

Final Fish Acute Value=4,900 μg/l
Final Invertebrate Acute Value=3,500 μg/l
Final Acute Value=3,500 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=220,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=220,000 μg/l
0.44×Final Acute Value=1,500 μg/l

*1.2.4-trichlorobenzene*

Final Fish Acute Value=470 μg/l
Final Invertebrate Acute Value=2,000 μg/l
Final Acute Value=470 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=35,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=35,000 μg/l
0.44×Final Acute Value=210 μg/l

*1.2.3.5-tetrachlorobenzene*

Final Fish Acute Value=900 μg/l
Final Invertebrate Acute Value=390 μg/l
Final Acute Value=390 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=17,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=17,000 μg/l
0.44×Final Acute Value=170 μg/l

*1.2.4.5-tetrachlorobenzene*

Final Fish Acute Value=220 μg/l
Final Invertebrate Acute Value=not available
Final Acute Value=220 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=47,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=47,000 μg/l
0.44×Final Acute Value=97 μg/l

*Pentachlorobenzene*

Final Fish Acute Value=36 μg/l
Final Invertebrate Acute Value=210 μg/l
Final Acute Value=36 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=6,600 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=6,600 μg/l
0.44×Final Acute Value=16 μg/l

*Saltwater Aquatic Life*

*1,2,4,5-tetrachlorobenzene.* The maximum concentration of 1,2,4,5-tetrachlorobenzene is the Final Acute Value of 26 μg/l and the 24-hour average concentration is the Final Chronic Value of 9.6 μg/l. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,2,4,5-tetrachlorobenzene the criterion to protect saltwater aquatic life as derived using the Guidelines is 9.6 μg/l as a 24-hour average and the concentration should not exceed 26 μg/l at any time.

No saltwater criterion can be derived for any other chlorinated benzene using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available.

However, data for 1,2,4,5-tetrachlorobenzene and saltwater organisms and 1,2-dichlorobenzene and freshwater organisms can be used as the basis for estimating criteria.

For 1,2,4,5-tetrachlorobenzene and saltwater organisms 0.44 times the Final Acute Value is 11 μg/l and this concentration is close to the Final Chronic Value of 9.6 μg/l derived from an embryo-larval test with the sheepshead minnow. Also, for 1,2-dichlorobenzene and freshwater organisms 0.44 times the Final Acute Value is less than the Final Chronic Value based on an embryo-larval test with the fathead minnow. Therefore, a reasonable estimate for other chlorinated benzenes and saltwater organisms would be 0.44 times the Final Acute Value.

*Chlorobenzene.* The maximum concentration of chlorobenzene is the Final Acute Value of 280 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For chlorobenzene the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 120 μg/l as a 24-hour average and the concentration should not exceed 280 μg/l at any time.

*1,2,4-trichlorobenzene.* The maximum concentration of 1,2,4-trichlorobenzene is the Final Acute Value of 7.8 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,2,4-trichlorobenzene the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 3.4 μg/l as a 24-hour average and the concentration should not exceed 7.8 μg/l at any time.

*1,2,3,5-tetrachlorobenzene.* The maximum concentration of 1,2,3,5-tetrachlorobenzene is the Final Acute Value of 5.9 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,2,3,5-tetrachlorobenzene the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 2.6 μg/l as a 24-hour average and the concentration should not exceed 5.9 μg/l at any time.

*Pentachlorobenzene.* The maximum concentration of pentachlorobenzene is the Final Acute Value of 2.9 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For pentachlorobenzene the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 1.3 μg/l as a 24-hour average and the concentration should not exceed 2.9 μg/l at any time.

*Summary of Available Data*

The concentrations below have been rounded to two significant figures.

*Chlorobenzene*

Final Fish Acute Value=1,600 μg/l
Final Invertebrate Acute Value=280 μg/l
Final Acute Value=280 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=340,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=340,000 μg/l
0.44×Final Acute Value=120 μg/l

*1.2.4-trichlorobenzene*

Final Fish Acute Value=3,200 μg/l
Final Invertebrate Acute Value=7.8 μg/l
Final Acute Value=7.8 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=8,800 μg/l

Residue Limited Toxicant Concentration=not available
Final Chronic Value=8,800 μg/l
0.44×Final Acute Value=3.4 μg/l

*1,2,3,5-tetrachlorobenzene*

Final Fish Acute Value=540 μg/l
Final Invertebrate Acute Value=5.9 μg/l
Final Acute Value=5.9 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=700 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=700 μg/l
0.44×Final Acute Value=2.6 μg/l

*1,2,4,5-tetrachlorobezene*

Final Fish Acute Value=120 μg/l
Final Invertebrate Acute Value=26 μg/l
Final Acute Value=26 μg/l
Final Fish Chronic Value=9.6 μg/l
Final Invertebrate Chronic Value=not available
Final Plant Value=7,100 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=9.6 μg/l
0.44×Final Acute Value=11 μg/l

*Pentachlorobenzene*

Final fish Acute Value=120 μg/l
Final Invertebrate Acute Value=2.9 μ/l
Final Acute Value=2.9 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=2,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=2,000 μg/l
0.44×Final Acute Value=1.3 μg/l

*Human Health*

*Monochlorobenzene.* There is no information in the literature which indicates that monochlorobenzene is, or is not, carcinogenic. There is enough evidence to suggest that MCB does cause dose related target organ toxicity, though the data still want for an acceptable chronic toxicity study. There is little, if any, usable human exposure data primarily because the exposure was not only to MCB but to other compounds of known toxicity.

The no observable adverse effect level (NOAEL) for derivation of the water quality criterion is derived from the information in the studies by Knapp, et al. (1971) and Irish (1963). These are 27.25 mg/kg/day for the dog (the next highest dose was 54.5 mg/kg and showed an effect), 12.5 mg/kg/rat from the Knapp study (the next highest dose was 50 mg/kg and showed an effect), and 14.4 mg/kg/rat from the Irish study (the next highest dose was 144 mg/kg and showed an effect). When toxic effects were observed at higher doses, the dog was judged to be somewhat more sensitive than rats. The Irish study ran over a period of six months which was twice as long as the Knapp study of both species. Since the Knapp and Irish studies appear to give similar results and since there are no chronic toxicities to rely on, it was decided to take the NOAEL level from the longest term study; that is, 14.4 mg/kg for six months.

Considering that there are relatively little human exposure data, that there is no long-term animal data, and that some theoretical questions, at least, can be raised on the possible effects of chlorobenzene on blood-forming tissue, it was decided to use an uncertainty factor of 1,000. From this the acceptable daily intake (ADI) can be calculated as follows:

$$\text{ADI} = \frac{70\text{ kg} \times 14.4\text{ mg/kg}}{1{,}000} = 1.008\text{ mg/day}$$

The average daily consumption of water was taken to be two liters and the consumption of fish to be 0.0187 kg daily. A bioconcentration factor of 13 was utilized. This is the value reported by the Duluth EPA Laboratories. The following calculation results in an acceptable criterion based on the available toxicologic data:

$$\frac{1.008}{2+(13\times 0.0187)} = 450\ \mu\text{g/l}$$

Varshavskya (1968), the only report available, has reported the threshold concentration for odor and taste of MCB in reservoir water as being 20 μg/l. This value is about 4.5 percent of the possible standard calculated above. It is, however, approximately 17 times greater than the highest concentration of MCB measured in survey sites.

Since water of disagreeable taste and odor is of significant influence on the quality of life, and thus, related to health, it would appear that the organoleptic level of 20 μg/l should be the recommended criterion.

*Trichlorobenzene.* While the committee recognizes a need for toxicological information in order to establish a criterion, there are no reliable published toxicological data on TCB. The studies by Smith, et al. (1978), and Coate, et al. (1977) do not give sufficient basis for establishing a toxicological criterion. Therefore, in lieu of a criterion based on toxicological information, an organoleptic level of 13 μg/l (Varshavskaya, 1968) is recommended. It should be emphasized that this is a criterion based on aesthetic rather than on health effects. Data on human health effects need to be developed as a more substantial basis for setting a criterion for the protection of human health.

*Tetrachlorobenzene.* The dose of 5 mg/kg/day reported for beagles (Braun, 1978) was utilized as the NOAEL for criterion derivation. An acceptable daily intake (ADI) can be calculated from the NOAEL by using a safety factor of 1,000 based on a 70 kg/man:

$$\text{ADI} = \frac{70\text{ kg} \times 5\text{ mg/kg}}{1{,}000} = 0.35\text{ mg/day}$$

For the sake of establishing a water quality criterion, it is assumed that on the average, a person ingests 2 liters of water and 18.7 gram of fish. Since fish may biomagnify this compound, a biomagnification factor (F) is used in the calculation.

The equation for calculating an acceptable amount of TeCB in water is:

$$\text{Criterion} = \frac{350\ \mu\text{g/day}}{2\text{ l}+(1{,}000\times 0.0187)} = 16.9\ \mu\text{g/l or } 17\ \mu\text{g/l}$$

Where:

2 l=2 liters of drinking water consumed
0.0187 kg=amount of fish consumed daily
1,000=biomagnification factor
ADI=Acceptable Daily Intake (mg/day for a 70 kg person)

Thus, the recommended criterion for TeCB in water is 17 μg/l.

*Pentachlorobenzene.* A survey of the QCB literature revealed no acute, subchronic or chronic toxicity data with the exception of the studies by Khera and Villeneuve (1975). These authors found an adverse effect on the fetal development of embryos exposed *in utero* to pentachlorobenzene. The adverse effect has not been labeled teratogenic because the abnormality was an increased incidence of extra ribs and sternal defects. The lowest level of exposure to the pregnant rat was 5 mg/kg. The criterion rationale is based on this exposure level. Since there was no no-observable-adverse effect level (NOAEL) an uncertainty factor of 5000 is used. The use of this factor has precedent in the pesticide literature.

From this, the acceptable daily intake (ADI) can be calculated as follows:

$$\text{ADI} = \frac{70\text{ kg} \times 5\text{ mg/kg}}{5{,}000} = 0.07\text{ mg}$$

The average daily consumption of water was taken to be 2 liters and the consumption of fish to be 0.0187 kg daily. The bioconcentration factor for QCB is 7,800.

Therefore:

$$\text{Recommended Criterion} = \frac{0.07}{2+(7{,}800)\times 0.0187} = .47\ \mu\text{g/l (or } 0.5\ \mu\text{g/l)}$$

The recommended water quality criterion for pentachlorobenzene is 0.5 μg/l.

*Hexachlorobenzene.* Among the studies reviewed by this document, only two appear suitable for use in the risk assessment: the mouse study of Cabral, et al. (1978) and the hamster study of Cabral, et al. (1977). These two studies are described in detail in Appendix I.

Under the Consent Decree in *NRDC* v. *Train*, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." HCB is suspected of being a human carcinogen. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of HCB in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be unfeasible in some cases, and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of HCB corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$, or $10^{-7}$ as shown in the table below:

| Exposure assumption (per day) | Risk levels and corresponding criteria [1] | | |
|---|---|---|---|
| | $10^{-7}$ (ng/l) | $10^{-6}$ (ng/l) | $10^{-5}$ (ng/l) |
| 2 liters of drinking water and consumption of 18.7 grams fish and shellfish[2] | 0.0125 | 0.125 | 1.25 |
| Consumption of fish and shellfish only | 0.0126 | 0.126 | 1.26 |

[1] Calculated by applying a modified "one-hit" extrapolation model described in the FEDERAL REGISTER, 44 FR 15926, March 15, 1979. Appropriate bioassay data used in the calculation of the model is presented in Summary of Pertinent Data. Since the extrapolation model is linear at low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1,000, and so forth.

[2] Ninety-nine percent of the HCB exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 12,000-fold. The remaining one percent of HCB exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of HCB, (1) occurring from the consumption of both drinking water and aquatic life grown in waters containing the corresponding HCB concentrations and, (2) occurring solely from consumption of aquatic life grown in the waters containing the corresponding HCB concentrations. Because data indicating other sources of HCB exposure and their contributions to total body burden are inadequate for quantitative use, the figures reflect the incremental risks associated with the indicated routes only.

**Summary of Recommended Criterion for Chlorinated Benzenes**

| Substance | Criterion | Basis for criterion |
|---|---|---|
| Monochlorobenzene [1] | 20μg/l | Organoleptic effects |
| Trichlorpbenzene | 13μg/l | Organoleptic effects |
| Tetrachlorobenzene | 17μg/l | Toxicity studies. |
| Pentachlorobenzene | .5μg/l | Toxicity study. |
| Hexachlorobenzene [2] | 1.25ng/l | Carcinogenicity. |

[1] A toxicological evaluation of monochlorobenzene resulted in a level of 450 μg/l; however, organoleptic effects have been reported at 20 μg/l.

[2] The value 1.25 ng/l is at a risk level of 1 in 100,000.

*Summary of Pertinent Data*

The water quality criterion for HCB is based on the induction of hepatomas and hemangioendotheliomas in male Syrian Golden hamsters given a daily oral dose of 100 ppm for 80 weeks (Cabral, et al. 1977). The hepatoma incidence was 26/30 in the treated group compared with 0/40 in the control group, and the hemangioendothelioma incidence was 6/30 in the treated group compared with 0/40 in the control group. The criterion was calculated from the following parameters.

$n^t$ hepatoma = 26
$N^t$ hepatoma = 30
$n^c$ hepatoma = 0
$N^c$ hepatoma = 40
$n^t$ hemangioendothelioma = 6
$N^t$ hemangioendothelioma = 30
$n^c$ hemangioendothelioma = 0
$N^c$ hemangioendothelioma = 40
Le = 80 wk
le = 80 wk
L = 80 wk
d = 100 ppm × 0.8 = 8 mg/kg/day
W = .100 kg
F = .0187 kg
R = 12,000

Based on these parameters, the one-hit slope ($B_H$) is 2.2363 $(mg/kg/day)^{-1}$ for hepatomas and 0.2477 $(mg/kg/day)^{-1}$ for hemangioendotheliomas. The resulting water concentration of HCB calculated to keep the individual lifetime cancer risk below $10^{-5}$ is 1.25 nanograms per liter.

**Chlorinated Ethanes**

*Criteria Summary*

*Freshwater Aquatic Life.* The data base for freshwater aquatic life is insufficient to allow use of the Guidelines. The following recommendation is inferred from toxicity data on pentachloroethane and saltwater organisms.

For 1,2-dichloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 3,900 μg/l as a 24-hour average and the concentration should not exceed 8,800 μg/l at any time.

For 1,1,1-trichloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 5,300 μg/l as a 24-hour average and the concentration should not exceed 12,000 μg/l at any time.

For 1,1,2-trichloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 310 μg/l as a 24-hour average and the concentration should not exceed 710 μg/l at any time.

For 1,1,1,2-tetrachloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 420 μg/l as a 24-hour average and the concentration should not exceed 960 μg/l at any time.

For 1,1,2,2-tetrachloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 170 μg/l as a 24-hour average and the concentration should not exceed 380 μg/l at any time.

For pentachloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 440 μg/l as a 24-hour average and the concentration should not exceed 1,000 μg/l at any time.

For hexachloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 62 μg/l as a 24-hour average and the concentration should not exceed 140 μg/l at any time.

For hexachloroethane the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 7.0 μg/l as a 24-hour average and the concentration should not exceed 16 μg/l at any time.

*Saltwater Aquatic Life.* The data base for saltwater aquatic life is insufficient to allow use of the Guidelines. The following recommendation is inferred from toxicity data on pentachloroethane and saltwater organisms.

For 1,2-dichloroethane the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 880 μg/l as a 24-hour average and the concentration should not exceed 2,000 μg/l at any time.

For 1,1,1-trichloroethane the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 240 μg/l as a 24-hour average and the concentration should not exceed 540 μg/l at any time.

For saltwater aquatic life, no criterion for 1,1,2-trichloroethane can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

For saltwater aquatic life, no criterion for 1,1,1,2-tetrachloroethane can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

For 1,1,2,2-tetrachloroethane the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 70 μg/l as a 24-hour average and the concentration should not exceed 160 μg/l at any time.

For pentachloroethane the criterion to protect saltwater aquatic life as derived using the Guidelines is 38 μg/l as a 24-hour average and the concentration should not exceed 87 μg/l at any time.

*Human Health.* For the maximum protection of human health from the potential carcinogenic effects of exposure to 1,2-dichloroethane, 1,1,2-trichloroethane, 1,1,2,2-tetrachloroethane and hexachloroethane through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of these chlorinated ethanes estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion Formulation section of this document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria as follows:

| Compound | Risk levels and corresponding criteria | | |
|---|---|---|---|
| | $10^{-5}$ μg/l | $10^{-6}$ μg/l | $10^{-7}$ μg/l |
| 1,2-dichloroethane | 7.0 | .70 | .07 |
| 1,1,2-trichloroethane | 2.7 | .27 | .027 |
| 1,1,2,2-tetrachloroethane | 1.8 | .18 | .018 |
| hexachloroethane | 5.9 | .59 | .059 |

For the protection of human health from the toxic properties of 1,1,1-trichloroethane ingested through the consumption of water and fish, the criterion is 15.7 mg/l.

At the present, there are insufficient data to derive criteria for monochloroethane, 1,1-dichloroethane, 1,1,1,2-tetrachloroethane and pentachloroethane.

*Basis for the Criteria*

*Freshwater Aquatic Life.* No freshwater criterion can be derived for any chlorinated ethane using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available.

However, data for pentachloroethane and saltwater organisms can be used as the basis for estimating criteria.

For pentachloroethane and saltwater organisms, 0.44 times the Final Acute Value is less than the Final Chronic Value derived from a life cycle test with the mysid shrimp. Therefore, a reasonable estimate of criteria for other chlorinated ethanes and freshwater organisms would be 0.44 times the Final Acute Value.

*1,2-dichloroethane.* The maximum concentration of 1,2-dichloroethane is the Final Acute Value of 8,800 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,2-dichloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 3,900 μg/l as a 24-hour average and the concentration should not exceed 8,800 μg/l at any time.

*1,1,1-trichloroethane.* The maximum concentration of 1,1,1-trichloroethane is the Final Acute Value of 12,000 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,1,1-trichloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 5,300 μg/l as a 24-hour average and the concentration should not exceed 12,000 μg/l at any time.

*1,1,2-trichloroethane.* The maximum concentration of 1,1,2-trichloroethane is the Final Acute Value of 710 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,1,2-trichloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 310 μg/l as a 24-hour average and the concentration should not exceed 710 μg/l at any time.

*1,1,1,2-tetrachloroethane.* The maximum concentration of 1,1,1,2-tetrachloroethane is the Final Acute Value of 960 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,1,1,2-tetrachloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 420 μg/l as a 24-hour average and the concentration should not exceed 960 μg/l at any time.

*1,1,2,2-tetrachloroethane.* The maximum concentration of 1,1,2,2-tetrachloroethane is the Final Acute Value of 380 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,1,2,2-tetrachloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 170 μg/l as a 24-hour average and the concentration should not exceed 380 μg/l at any time.

*Pentachloroethane.* The maximum concentration of pentachloroethane is the Final Acute Value of 1,000 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For pentachloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 440 μg/l as a 24-hour average and the concentration should not exceed 1,000 μg/l at any time.

Hexachloroethane. The maximum concentration of hexachloroethane is the Final Acute Value of 140 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For hexachloroethane the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 62 μg/l as a 24-hour average and the concentration should not exceed 140 μg/l at any time.

*Summary of Available Data*

The concentrations below have been rounded to two significant figures.

*1,2-dichloroethane*

Final Fish Acute Value=68,000 μg/l
Final Invertebrate Acute Value=8,800 μg/l
Final Acute Value=8,800 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=not available
Residue Limited Toxicant Concentration=not available
Final Chronic Value=not available
0.44 × Final Acute Value=3,900 μg/l

*1,1,1-trichloroethane*

Final Fish Acute Value=12,000 μg/l
Final Invertebrate Acute Value=not available
Final Acute Value=12,000 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=not available
Residue Limited Toxicant Concentration=not available
Final Chronic Value=not available
0.44 × Final Acute Value=5,300 μg/l

*1,1,2-trichloroethane*

Final Fish Acute Value=5,700 μg/l
Final Invertebrate Acute Value=710 μg/l
Final Acute Value=710 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=not available
Residue Limited Toxicant Concentration=not available
Final Chronic Value=not available
0.44 × Final Acute Value=310 μg/l

*1,1,1,2-tetrachloroethane*

Final Fish Acute Value=2,700 μg/l
Final Invertebrate Acute Value=960 μg/l
Final Acute Value=960 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=not available
Residue Limited Toxicant Concentration=not available
Final Chronic Value=not available
0.44 × Final Acute Value=420 μg/l

*1,1,2,2-tetrachloroethane*

Final Fish Acute Value=3,000 μg/l
Final Invertebrate Acute Value=380 μg/l
Final Acute Value=380 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=140,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=140,000 μg/l
0.44 × Final Acute Value=170 μg/l

*Pentachloroethane*

Final Fish Acute Value=1,000 μg/l
Final Invertebrate Acute Value=2,500 μg/l
Final Acute Value=1,000 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=120,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=120,000 μg/l
0.44 × Final Acute Value=440 μg/l

*Hexachloroethane*

Final Fish Acute Value=140 μg/l
Final Invertebrate Acute Value=330 μg/l
Final Acute Value=140 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=87,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=87,000 μg/l
0.44 × Final Acute Value=62 μg/l

*Saltwater Aquatic Life*

*Pentachloroethane.* The maximum concentration of pentachloroethane is the Final Acute Value of 87 μg/l and the 24-hour average concentration is the Final Chronic Value of 38 μg/l. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For pentachloroethane the criterion to protect saltwater aquatic life as derived using the Guidelines is 38 μg/l as a 24-hour average and the concentration should not exceed 87 μg/l at any time.

No saltwater criteria can be derived for other chlorinated ethanes using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available.

However, data for pentachloroethane and saltwater organisms can be used as the basis for estimating criteria.

For pentachloroethane and saltwater organisms, 0.44 times the Final Acute Value is less than the Final Chronic Value derived from a life cycle test with the mysid shrimp. Therfore, a reasonable estimate of criteria for other chlorinated ethanes and saltwater organisms would be 0.44 times the Final Acute Value.

*1,2-dichloroethane.* The maximum concentration of 1,2-dichloroethane is the Final Acute Value of 2,000 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,2-dichloroethane the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 880 μg/l as a 24-hour average and the concentration should not exceed 2,000 μg/l at any time.

*1,1,1—trichloroethane.* The maximum concentration of 1,1,1-trichloroethane is the Final Acute Value of 540 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,1,1-trichloroethane the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 240 μg/l as a 24-hour average and the concentration should not exceed 540 μg/l at any time.

*1,1,2,2-tetrachlorethane.* The maximum concentration of 1,1,2,2-tetrachloroethane is the final Acute Value of 160 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For 1,1,2,2-tetrachloroethane the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 70 μg/l as a 24-hour average and the concentration should not exceed 160 μg/l at any time.

*Hexachloroethane.* The maximum concentration of hexachloroethane is the Final Acute Value of 16 μg/l and the estimated 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For hexachloroethane the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 7.0 μg/l as a 24-hour average and the concentration should not exceed 16 μg/l at any time.

*Summary of Available Data*

The concentrations below have been rounded to two significant figures.

*1,2,-dichloroethane*

Final Fish Acute Value=not available
Final Invertebrate Acute Value=2,000 μg/l
Final Acute Value=2,000 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=greater than 433,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=greater than 433,000 μg/l
0.44×Final Acute Value=880 μg/l

*1,1,1-trichloroethane*

Final Fish Acute Value=10,000 μg/l
Final Invertebrate Acute Value=540 μg/l
Final Acute Value=540 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=greater than 669,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=greater than 669,000 μg/l
0.44×Final Acute Value=240 μg/l

*1,1,2,2-tetrachloroethane*

Final Fish Acute Value=1,800 μg/l
Final Invertebrate Acute Value=160 μg/l
Final Acute Value=160 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=6,200 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=6,200 μg/l
0.44×Final Acute Value=70 μg/l

*Pentachloroethane*

Final Fish Acute Value=17,000 μg/l
Final Invertebrate Acute Value=87 μg/l
Final Acute Value=87 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=110 μg/l
Final Plant Value=58,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=110 μg/l
0.44×Final Acute Value=38 μg/l

*Hexachloroethane*

Final Fish Acute Value=350 μg/l
Final Invertebrate Acute Value=16 μg/l
Final Acute Value=16 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=7,800 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=7,800 μg/l
0.44×Final Acute Value=7.0 μg/l

*Human Health*

**Table I.—*Criteria for Chloroethanes***

| Compound | Criterion | Reference |
|---|---|---|
| Monochloroethane | None | |
| 1,1-Dichloroethane | None | |
| 1,1-Dichloroethane | 7.0 μg/l—Carcinogenicity data | NCI, 1978a |
| 1,1,1-Trichloroethane | 15.7 mg/l—Mammalian toxicity data | NCI, 1977 |
| 1,1,2-Trichloroethane | 2.7 μg/l—Carcinogenicity data | NCI, 1978b |
| 1,1,1,2-Tetrachloroethane | None | |
| 1,1,2,2-Tetrachloroethane | 1.8 μg/l—Carcinogenicity data | NCI, 1978c |
| Pentachloroethane | None | |
| Hexachloroethane | 5.9 μg/l—Carcinogenicity data | NCI, 1978d |

At the present time, there is insufficient mammalian toxicological information to establish a water criterion for human health for the following chloroethanes: monochloroethane, 1,1-dichloroethane, 1,1,1,2-tetrachloroethane and pentachloroethane. Available evidence indicates that the general population is exposed to only trace levels of 1,1-dichloroethane, 1,1,1,2-tetrachloroethane and pentachloroethane. Although inhalation exposure to monochloroethane is more widespread, it is considered one of the least toxic of the chloroethanes. Should significant levels of exposure be documented in the future, it will be necessary to conduct more extensive toxicologic studies with these chloroethanes.

The criterion for 1,1,1-trichloroethane is based on the National Cancer Institute bioassay for possible carcinogenicity (1977). Results of the study showed that the survival of both Osborne-Mendel rats and B6C3F1 mice was significantly decreased in groups receiving oral doses of 1,1,1-trichloroethane. Chronic murine pneumonia may have been responsible for the high incidence of natural deaths. A variety of neoplasms was observed in both species, however, the incidence of specific malignancies was not significantly different from those observed in control animals. Survival time was significantly decreased in rats receiving the high dose, therefore, the criterion for 1,1,1-trichloroethane is based on the low dose in rats (750 mg/kg body weight, 5 days/week for 78 weeks) which produced toxic effects in a number of systems. It should be recognized that the actual no-observable-adverse-effect level (NOAEL) will be lower. However, use of the lowest-minimal-effect dose as an estimate of an "acceptable daily intake" has been practiced by the National Academy of Sciences (1977). Thus, assuming a 70 kg body weight and using a safety factor of 1,000 (Natl. Acad. Sci., 1977) the following calculation can be derived:

$$\frac{750\ \text{mg/kg} \times 70\ \text{kg} \times 5/7\ \text{day}}{1000} = 37.5\ \text{mg/day}$$

Therefore, consumption of 2 liters of water daily and 18.7 grams of contaminated fish having a bioconcentration factor of 21, would result in, assuming 100 percent gastrointestinal absorption of 1,1,1-trichloroethane, a maximum permissible concentration of 15.7 mg/l for ingested water:

$$\frac{37.5\ \text{mg/day}}{2\ \text{liters} + (21 \times .0187) \times 1.0} = 15.7\ \text{mg/l}$$

Based on available literature, 1,1,2-tri-, 1,1,2,2-tetra-, and hexachloroethane are considered to be carcinogenic in at least one rodent species (Natl. Cancer Inst., 1978b,c,d). In the case of these three chloroethanes, a statistical evaluation of the incidences of hepatocellular carcinomas revealed a significant positive association between the administration of the respective chloroethanes and tumor incidence. It can be concluded that under the conditions of the NCI bioassay, 1,1,2-tri; 1,1,2,2-tetra-; and hexachloroethane are carcinogenic in B6C3F1 mice, inducing (in all cases) hepatocellular carcinomas in either male or female mice.

Estimated risk levels for these chloroethanes in water can be calculated using a linear, non-threshold model with the results from the NCI bioassays (see Summary of Pertinent Data). The model assumes a risk of 1 in 100,000 of developing cancer as a result of drinking 2 liters of water per day containing chloroethane at the concentrations used in the bioassays. Allowances are also made for consuming fish from chloroethane contaminated waters. Based upon these assumptions, the following criteria can be calculated:

| Chloroethane | Dose[a] (mg/kg) | Criteria (μg/l) |
|---|---|---|
| 1,1,2-trichloroethane | 279 | 2.7 |
| 1,1,2,2-tetrachloroethane | 203 | 1.8 |
| hexachloroethane | [illegible] | 4.4 |

[a]Five days per week for 78 weeks.

Under the conditions of an NCI bioassay (1978a) 1,2-dichloroethane is carcinogenic, inducing a statistically significant number of squamous cell carcinomas of the forestomach and hemangiosarcomas of the circulatory system in male rats, mammary adenocarcinomas in female rats and mice, and endometrial tumors in female mice. The criterion for 1,2-dichloroethane is based on the high dose (107 mg/kg/body weight, 5 days/week for 78 weeks) which induced mammary

adenocarcinomas in female rats. Using a linear, non-threshold model and including the consumption of fish from chloroethane contaminated waters the criterion for 1,2-dichloroethane is 7.0 μg/l.

It must be recognized that the NCI studies were designed to provide a "yes/no" answer to the carcinogenicity of a chemical in rats and mice. In some cases, it is difficult to justify extrapolation of data from NCI studies in order to assess the risk to man of chronic exposure to low concentrations of a chemical. Those who assess risk should be aware of the following: Impurities in technical grade chloroethanes were not identified; chloroethanes were administered in oil which may affect absorption and metabolism; high concentrations were used; a time-weighted average dose was reported; however, doses causing toxic responses were often administered cyclically (one week, no treatment, followed by four weeks of treatment, five days/week); during some experiments dose levels were lowered or raised; for criteria calculations, doses administered five days/week were adjusted to an average daily dose as if administered seven days/week.

Under the Consent Decree in NRDC vs. Train, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." 1,2-Dichloroethane, 1,1,2-trichloroethane, 1,1,2,2,-tetrachloroethane and hexachloroethane are suspected of being human carcinogens. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of these chlorinated ethanes in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assit the Agency and States in the possible future development of water quality regulations, the concentrations of these chlorinated ethanes corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$ or $10^{-7}$ as shown in the table below.

| Exposure assumptions | Risk levels and corresponding criteria[1] | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ μg/l | $10^{-6}$ μg/l | $10^{-5}$ μg/l |
| 2 liters of drinking water and consumption of 18.7 grams of fish and shellfish:[2] | | | | |
| 1,2-dichloroethane | 0 | 0.07 | 0.07 | 7.0 |
| 1,1,2-trichloroethane | 0 | 0.027 | 0.27 | 2.7 |
| 1,1,2,2-tetrachloroethane | 0 | 0.018 | 0.18 | 1.8 |
| hexachloroethane | 0 | 0.059 | 0.59 | 5.9 |
| Consumption of fish and shellfish only: | | | | |
| 1,2-dichloroethane | 0 | 1.708 | 17.08 | 170.8 |
| 1,1,2-trichloroethane | 0 | 0.483 | 4.83 | 48.3 |
| 1,1,2,2-tetrachloroethane | 0 | 0.127 | 1.27 | 12.7 |
| hexachloroethane | 0 | 0.079 | 0.79 | 7.9 |

[1] Calculated by applying a modified "one hit" extrapolation model described in the 44 FR 15926, March 15, 1979. Appropriate bioassay data used in the calculation of the model are presented in the summary of pertinent data. Since the extrapolation model is linear to low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1,000, and so forth.

[2] Four percent of 1,2-dichloroethane exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 4.6 fold. The remaining 96 percent of 1,2-dichloroethane exposure results from drinking water.

Six percent of 1,1,2-trichloroethane exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 6.3 fold. The remaining 94 percent of 1,1,2-trichloroethane exposure results from drinking water.

Fourteen percent of 1,1,2,2-tetrachloroethane exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 18 fold. The remaining 86 percent of 1,1,2,2-tetrachloroethane exposure results from drinking water.

Seventy-five percent of hexachloroethane exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potentnial of 320 fold. The remaining 25 percent of hexachloroethane exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of these chlorinated ethanes (1) occurring from the consumption of both drinking water and aquatic life grown in water containing the corresponding chlorinated ethane concentrations and, (2) occurring solely from the consumption of aquatic life grown in the waters containing the corresponding chlorinated ethane concentrations.

Although total exposure information for the above chlorinated ethanes is discussed and an estimate of the contributions from other sources of exposure can be made, this data will not be factored into the ambient water quality criteria formulation because of the tenuous estimates. The criteria presented, therefore, assume an incremental risk from ambient water exposure only.

*Summary of Pertinent Data for 1,2-Dichloroethane*

The water quality criterion for 1,2-dichloroethane is based on the induction of mammary adenocarcinomas in female Osborne-Mendel rats, given an average oral dose of 107 mg/kg/day 1,2-dichloroethane over a period of 78 weeks (NCI, 1978a). The incidences of mammary adenocarcinomas were 18/50 and 0/20 in the treated and control groups, respectively. The criterion was calculated from the following parameters:

$n_t = 18$
$N_t = 50$
$n_c = 0$
$N_c = 20$
$Le = 110$ wks.
$le = 69$ wks.
$L = 110$ wks.
$d = 76.4$ mg/kg/day (107 mg/kg/day $\times 5/7$)
$f = 0.0187$ kg/day
$R = 4.6$
$w = 0.319$ kg

Based on these parameters, the one-hit slope ($B_H$) is 0.04765 $(mg/kg/day)^{-1}$. The concentration of 1,2-dichloroethane in water, calculated to keep the lifetime cancer risk below $10^{-5}$ is 7.0 μg/l.

*Summary of Pertinent Data for 1,1,2-trichloroethane*

The water quality criterion for 1,1,2-trichloroethane is based on the induction of hepatocellular carcinomas in female B6C3F1 mice, given an average oral dose of 390 mg/kg/day over a 78

week period (NCI, 1978b). The incidences of hepatocellular were 40/45 and 0/20 in the treated and control groups, respectively. The criterion was calculated from the following parameters:

$n_t = 40$
$N_t = 45$
$n_c = 0$
$N_c = 20$
Le = 91 wks.
le = 78 wks.
L = 91 wks.
d = 279 mg/kg/day (390 mg/kg/day × 5/7)
F = 0.0187 kg/day
R = 6.3
w = 0.029 kg

Based on these parameters, the one-hit slope ($B_H$) is 0.123 $(mg/kg/day)^{-1}$. The concentration of 1,1,2-trichloroethane in water, calculated to keep the lifetime cancer risk below $10^{-5}$ is 2.7 μg/l.

*Summary of Pertinent Data for 1,1,2,2-Tetrachloroethane*

The water quality criterion for 1,1,2,2-tetrachloroethane is based on the induction of hepatocellular carcinomas in male B6C3F1 mice, receiving average oral doses of 284 mg/kg/day over a 78-week period (NCI, 1978c). The incidences of hepatocellular carcinomas were 4¾9 and 1/18 in the treated and control groups, respectively. The criterion was calculated from the following parameters:

$n_t = 44$
$N_t = 49$
$n_c = 1$
$N_c = 18$
Le = 91 wks
le = 78 wks
L = 91 wks
d = 203 mg/kg/day (284 mg/kg/day × 5/7)
F = 0.0187 kg/day
R = 18
w = 0.035 kg

Based on these parameters, the one-hit slope ($B_H$) is 0.1638 $(mg/kg/day)^{-1}$. The concentration of 1,1,2,2-tetrachloroethane in water, calculated to keep the lifetime cancer risk below $10^{-5}$, is 1.8 μg/l.

*Summary of Pertinent Data for Hexachloroethane*

The water quality criterion for hexachloroethane is based on the induction of hepatocellular carcinomas in male B6C3F1 mice, given an average oral dose of 1,179 mg/kg/day over a 78-week period (NCI, 1978d). The incidences of hepatocellular carcinomas were 3¼9 and 3/20 in the treated and control groups, respectively. The criterion was calculated from the following parameters:

$n_t = 31$
$N_t = 49$
$n_c = 3$
$N_c = 20$
Le = 91 wks
le = 78 wks
L = 91 wks
d = 842 mg/kg/day (1179 mg/kg/day × 5/7)
F = 0.0187 kg/day
R = 320
w = 0.032 kg

Based on these parameters, the one-hit slope ($B_H$) is 0.0149 $(mg/kg/day)^{-1}$. The concentration of hexachloroethane in water, calculated to keep the lifetime cancer risk below $10^{-5}$, is 5.9 μg/l.

**Chromium**

*Criteria Summary*

*Freshwater Aquatic Life.* For trivalent chromium the criterion to protect freshwater aquatic life as derived using the Guidelines is "e(0.83·ln (hardness)+2.94)" as a 24-hour average and the concentration should not exceed "e(0.83·ln(hardness)+3.72)" at any time.

For hexavalent chromium the criterion to protect freshwater aquatic life as derived using the Guidelines is 10 μg/l as a 24-hour average concentration and the concentration should not exceed 110 μg/l at any time.

*Saltwater Aquatic Life.* For saltwater aquatic life, no criterion for trivalent chromium can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

For hexavalent chromium the criterion to protect saltwater aquatic life as derived using the Guidelines is 25 μg/l as a 24-hour average and the concentration should not exceed 230 μg/l at any time.

*Human Health.* For the protection of human health from the toxic properties of chromium (except hexavalent chromium) ingested through water and contaminated aquatic organisms, the recommended water quality criterion is 50 μg/l.

For the maximum protection of human health from the potential carcinogenic effects of exposure to hexavalent chromium through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of hexavalent chromium estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion Formulation section of this document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria of 8 ng/l, 0.8 ng/l, and .08 ng/l, respectively.

*Basis for the Criteria*

Freshwater Aquatic Life

*Hexavalent chromium.* The maximum concentration of hexavalent chromium is the Final Acute Value of 110 μg/l and the 24-hour average concentration is the final Chronic Value of less than 10 μg/l. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For hexavalent chromium the criterion to protect freshwater aquatic life as derived using the Guidelines is 10 μg/l as a 24-hour average and the concentration should not exceed 110 μg/l at any time.

*Trivalent chromium.* The maximum concentration of trivalent chromium is the Final Acute Value of e(0.83·ln (hardness)+3.72) and the 24-hour average concentration is the Final Chronic Value of e(0.83·ln (hardness)+2.94). No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For trivalent chromium the criterion to protect freshwater aquatic life as derived using the Guidelines is "e(0.83·ln (hardness)+2.94)" as a 24-hour average and the concentration should not exceed "e(0.83·ln (hardness)+3.72)" at any time.

*Summary of Available Data*

The concentrations below have been rounded to two significant figures. All concentrations herein are expressed in terms of chromium.

*Hexavalent chromium*

Final Fish Acute Value = 13,079 μg/l
Final Invertebrate Acute Value = 110 μg/l
Final Acute Value = 110 μg/l
Final Fish Chronic Value = 26 μg/l
Final Invertebrate Chronic Value = less than 10 μg/l
Final Plant Value = 10 μg/l
Residue Limited Toxicant Concentration = not available
Final Chronic Value = less than 10 μg/l
0.44 × Final Acute Value = 48 μg/l

*Trivalent chromium*

Final Fish Acute Value = e(0.83·ln (hardness)+4.45)
Final Invertebrate Acute Value = e(0.83·ln (hardness)+3.72)
Final Acute Value = e(0.83·ln (hardness)+3.72)
Final Fish Chronic Value = not available
Final Invertebrate Chronic Value = e(0.83·ln (hardness)+2.94)
Final Chronic Value = e(0.83·ln (hardness)+2.94)
Final Plant Value = not available
Residue Limited Toxicant Concentration = not available

*Saltwater Aquatic Life.* The maximum concentration of hexavalent chromium is the Final Acute Value of 260 μg/l and the 24-hour average concentration is the Final Chronic Value of 25 μg/l. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

For hexavalent chromium the criterion to protect saltwater aquatic life as derived using the Guidelines is 25 μg/l as a 24-hour average and the concentration should not exceed 230 μg/l at any time.

For saltwater aquatic life, no criterion for trivalent chromium can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Summary of Available Data*

The concentrations below have been rounded to two significant figures. All concentrations herein are expressed in terms of chromium.

*Hexavalent chromium*

Final Fish Acute Value=7,800 μg/l
Final Invertebrate Acute Value=230 μg/l
Final Acute Value=230 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=25 μg/l
Final Plant Value=1,000 μg/l
Residue Limited Toxicant
Concentration=not available
Final Chronic Value=25 μg/l
0.44×Final Acute Value=100 μg/l

*Human Health.* There is evidence which suggests that hexavalent chromium (Cr VI) is a carcinogen. Based on exposure of chromium workers to Cr VI (Mancuso and Hueper, 1951; Taylor, 1966, the U.S. EPA Carcinogen Assessment Group has developed a water quality criterion for Cr VI to keep the lifetime risk level below one in 100,000 (see Appendix I).

Under the Consent Decree in NRDC vs. Train, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." Chromium VI is suspected of being a human carcinogen. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of Chromium VI in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of Chromium VI corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$ or $10^{-7}$ as shown in the table below.

| Exposure assumptions | Risk levels and Corresponding Criteria[1] | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ (ng/l) | $10^{-6}$ (ng/l) | $10^{-5}$ (ng/l) |
| 2 liters of drinking water and consumption of 18.7 grams of fish and shellfish[2] | | 0.08 | 0.8 | 8 |
| Consumption of fish and shellfish only | | [illegible] | 86.3 | 863 |

[1] Calculated by applying a modified "one hit" extrapolation model described in the FR 15926, 1979 to the animal bioassay data presented in the summary of pertinent data. Since the extrapolation model is linear to low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1,000, and so forth.

[2] Approximately one percent of the Chromium VI exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 1.0 fold. The remaining 99 percent of Chromium VI exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of Chromium VI (1) occurring from the consumption of both drinking water and aquatic life grown in water containing the corresponding Chromium VI concentrations and, (2) occurring solely from the consumption of aquatic life grown in the waters containing the corresponding Chromium VI concentrations. Although total exposure information for Chromium VI is discussed and an estimate of the contributions from other sources of exposure can be made, this data will not be factored into the ambient water quality criteria. The criteria presented, therefore, assume an incremental risk from ambient water exposure only.

Therefore, the criterion for hexavalent chromium should be at a level of no greater than 8 ng/l to keep the lifetime risk of cancer below 1 in 100,000.

A water quality criterion can be set for other Cr species on the basis of reasonable safety margins applied to the lowest exposure observed to produce effects.

The level of 0.05 mg/l of chromium quoted in Table 2 appears to be an acceptable risk level. This level is 500 times lower than a concentration which remained without overt toxicological effects in rats over a period of one year, and over 200 times lower than a level reported not to affect dogs over four years. With the exception of hexavalent chromium, there is no reason to believe that the level of 0.05 mg/l (50 μg/l) permitted for ambient water poses a significant threat to human health. As a standard, this level was set in 1962 and has in the meantime been confirmed by several reviewing groups. Therefore, the recommended water quality criterion for chromium, except hexavalent chromium, is 50 μg/l. For practical purposes, it should be noted that it is difficult to analytically distinguish between trivalent and hexavalent chromium.

Because of the low bioconcentration of chromium, consideration of the consumption of fish and shellfish does not change the recommended criterion:

If two liters of drinking water are ingested per day, then a level of 50 μg/l would correspond to an intake of 100 μg from water. To apportion this daily intake to both drinking water and fish and shellfish consumed, the following calculation can be used:

$2X+(0.0187)(F)(X)=100\ \mu g$

Where:

2=amount of water ingested in liter/day
X=chromium concentration in water, mg/1
0.0187=amount of fish consumed per day, kg/day
F=bioconcentration factor, mg chromium/kg fish per mg chromium in water. (F=11 for chromium)

$2X+.2X=100\mu g$
$2.2X=100\ \mu g$
$X=45\ \mu g/l$ (or ~ 50 μg/l)

*Summary of Pertinent Data*

In order to calculate a water quality criterion for Cr VI, it was necessary to assume that the population's exposure to Cr VI in the Mancuso and Hueper study was the same as the exposure in Taylor's paper. Taylor's is the only study in which the cohort is large enough (1212 people were studied) to see the effects of Cr exposure in areas other than the lungs, which are directly affected by inhaled Cr. The lung cancer risk was very high in this study. The risk of digestive cancer from Cr exposure is statistically significant in Taylor's cohort (as shown in 1974 by Enterline); however, the amount of Cr to which the workers were exposed is not available for Taylor's study. Mancuso and Hueper closely studied 97 chromium workers in which they saw a high incidence of lung cancer (however, less than Taylor's study). The data on exposure in the Mancuso and Hueper study is very detailed, giving information on first exposure date, years of exposure, latent

period, amount of Cr exposure in mg Cr/$m^3$ for Cr III and Cr VI separately, and date of death.

In order to calculate a water quality criterion for Cr, it is necessary to know the exposure levels producing the digestive cancer response in Taylor's study, as the direct lung effects may not be relevant to water exposure.

The following is an account of the calculations used in estimating the water concentration of Cr VI which would result in a lifetime risk of dying from digestive cancer of $10^{-5}$.

Assuming that the average exposure in Mancuso and Hueper's study is 0.1 mg Cr/$m^3$ (this is the mean exposure to water soluble chromium which is Cr VI), then the concentration in Taylor's study is also assumed to be 0.15 mg Cr/$m^3$. The total exposure in 4.146 years (the mean exposure time in Taylor's study) is 0.15 mg Cr/$m^3$ × 10 $m^3$/working day × 240 working days/yr × 4.146 years = 1492.56 mg. If 50 percent of this is swallowed from the respiratory tract, then 2.018 liter/day × 365 days/year × 70 years × C mg/l = 1492.56 × 0.5 (The bioconcentration factor in fish is 1.0)

C = 14.40 μg/l of Cr VI

C is the estimated concentration in water necessary to produce the observed digestive cancer incidence in the Taylor study. The relative risk in the Taylor study is 1.533 which is statistically significant. The excessive risk corresponding to a concentration of C = 14.40 μ/l is .533 p, where p is the expected population risk of digestive tract cancer. The slope of the excessive risk curve is

$$B = \frac{0.533p}{0.014} \quad 37.01p \text{ (mg/l)}^{-1}$$

The water quality criterion corresponding to a risk of $10^{-5}$ is given by

$$X = \frac{10^{-5}}{37.01p} \text{ mg/l}$$

$$= \frac{10^{-2}}{37.01p} \text{ μ/l}$$

Based on the HEW Vital Statistics of the United States (1973), the lifetime risk of dying from digestive cancer (p) is estimated by an actuarial method to be 3.5 percent.* Therefore, the water concentration of Cr VI should be less than 8.0 ng/l in order to keep the lifetime risk below $10^{-5}$.

Using the water concentration of 8 mg/l for Cr VI, the one-hit slope ($B_H$) may be calculated as follows:

$$B_H = \frac{70 \times 10^{-5}}{C(2 + R \times F)}$$

R = 1.0
F = .0187 kg/day
C = $8 \times 10^{-6}$ mg/l
$B_H$ = 43.345 (mg/kg/day)$^{-1}$

*(Thus, from this data, p = .035).

## DDT

*Criteria Summary*

*Freshwater Aquatic Life.* For DDT and metabolites the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.00023 μg/l as a 24-hour average and the concentration should not exceed 0.41 μg/l at any time.

*Saltwater Aquatic Life.* The data base for saltwater aquatic life is insufficient to allow use of the Guidelines. The following recommendation is inferred from toxicity data for freshwater organisms.

For DDT and metabolites the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 0.0067 μg/l as a 24-hour average and the concentration should not exceed 0.021 μg/l at any time.

*Human Health.* For the maximum protection of human health from the potential carcinogenic effects of exposure to DDT through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of DDT estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria of 0.98 ng/l, 0.098 ng/l, and .0098 ng/l, respectively. If water alone is consumed, the water concentration should be less than 0.36 μg/l to keep the lifetime cancer risk below $10^{-5}$.

*Basis for the Criteria*

*Freshwater Aquatic Life.* The maximum concentration of DDT and metabolites is the Final Acute Value of 0.41 μg/l and the 24-hour average concentration is the Final Chronic Value of 0.00023 μg/l. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

*Summary of Available Data.* The concentrations below have been rounded to two significant figures.

Final Fish Acute Value = 1.3 μg/l
Final Invertebrate Acute Value = 0.41 μg/l
Final Acute Value = 0.41 μg/l
Final Fish Chronic Value = 0.11 μg/l
Final Invertebrate Chronic Value = not available
Final Plant Value = 0.30 μg/l
Residue Limited Toxicant Concentration = 0.00023 μg/l
Final Chronic Value = 0.00023 μg/l
0.44 × Final Acute Value = 0.18 μg/l

*Saltwater Aquatic Life.* No saltwater criterion can be derived for DDT and metabolites using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available.

Results obtained with DDT and metabolites and freshwater organisms indicate how a criterion may be estimated for DDT and metabolites and saltwater organisms.

For DDT and metabolites and freshwater organisms the Residue Limited Toxicant Concentration is lower than the Final Fish Chronic Value which is derived from results of a life cycle test with the fathead minnow. Therefore, it seems reasonable to estimate a criterion for DDT and metabolites and saltwater organisms using the Residue Limited Toxicant Concentration as the Final Chronic Value.

The maximum concentration of DDT and metabolites is the Final Acute Value of 0.021 μg/l and the estimated 24-hour average concentration is the Final Chronic Value of 0.0067 μg/l. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

*Summary of Available Data.* The concentrations below have been rounded to two significant figures.

Final Fish Acute Value = 0.38 μg/l
Final Invertebrate Acute Value = 0.021 μg/l
Final Acute Value = 0.021 μg/l
Final Fish Chronic Value = not available μg/l
Final Invertebrate Chronic Value = not available
Final Plant Value = 10 μg/l
Residue Limited Toxicant Concentration = 0.0067 μg/l
Final Chronic Value = 0.0067 μg/l
0.44 × Final Acute Value = 0.0092 μg/l

*Human Health.* Since no epidemiological evidence for the carcinogenicity of DDT in man has been reported, the results of animal carcinogenicity studies conducted by feeding DDT or its metabolites over the lifespan of the animal are regarded as the most pertinent data. Although a number of studies have been reported for various species, the major evidence for the tumorigenicity of DDT is its ability to induce liver tumors in mice.

Under the Consent Decree in *NRDC vs. Train*, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." DDT

is suspected of being a human carcinogen. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of DDT in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of DDT corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additonal incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$ or $10^{-7}$ as shown in the table below.

| Exposure assumptions (per day) | Risk levels and corresponding criteria[1] | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ (ng/l) | $10^{-6}$ (ng/l) | $10^{-5}$ ng/l) |
| 2 liters of drinking water and consumption of 18.7 grams of fish and shellfish[2] | ..... | 0.0098 | 0.098 | 0.98 |
| Consumption of fish and shellfish only | ..... | 0.0098 | 0.098 | 0.98 |

[1] Calculated by applying a modified "one hit" extrapolation model described in the FR 15926, 1979, to the animal bioassay data presented in the summary of pertinent data. Since the extrapolation model is linear at low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1,000, and so forth.

[2] Greater than 99 percent of the DDT exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 39,000 fold. The remaining less than one percent of DDT exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of DDT (1) occurring from the consumption of both drinking water and aquatic life grown in water containing the corresponding DDT concentrations and, (2) occurring solely from the consumption of aquatic life grown in the waters containing the corresponding DDT concentrations. Although total exposure information for DDT is discussed and an estimate of the contributions from other sources of exposure can be made, this data will not be factored into the ambient water quality criteria formulation because of the tenuous estimates. The criteria presented, therefore, assume an incremental risk from ambient water exposure only.

The case of DDT and its possible role as a human carcinogen is complicated by several factors. Despite widespread use and exposure over thirty years, no positive associations with human cancer have been found to date, although the number of individuals studied is not statistically large. It is a chemical with high efficacy and has been extremely effective all over the world for public health measures. However, its slow biodegradability and propensity to accumulate in nontarget species have made it particularly hazardous for many fish and bird species. For mammals, however, it has a low acute toxicity as compared to other alternate pesticides.

DDT has not been shown to produce point mutations or teratogenic effects in a wide battery of tests. Some evidence for its clastogenic properties, however, make it suspect. The primary evidence for the carcinogenicity of DDT and metabolites to date has been the induction of liver tumors in mice. Studies in other species have consistently shown little or no effect and in the mice only liver tumors have shown an increase. The evidence for the carcinogenicity of DDT would be much more convincing if other species or sites of tumorigenic action could be conclusively demonstrated. This is in light of the fact that DDT has been probably the most extensively studied compound in modern science.

Current levels of exposure would seem to pose extremely small risk to persons in the U.S. DDT and DDE are preferentially stored in fatty compartments that are not actively dividing and subject to carcinogenic changes.

The use of DDT has been restricted in several countries because of its impact on the environment and its tumorigenic effect in mice. This is a reasonable proposition based on numerous reports. Therefore, the levels proposed in this document should ensure the health of wildlife and the co-existing human population.

*Summary of Pertinent Data.* The water quality criterion for DDT can be derived on the basis of two independent sets of data, neither of which is completely satisfactory. The first method is based on the most sensitive animal chronic bioassay available, which is the six-generation study by Turusov, et al. (Jour. Nat. Cancer Inst., 1973) in CF–1 mice. The second method is based on a comparison of the lifetime incidence of nervous system cancer cases between residents of New York State (except New York City) and residents of Israel who were born in Europe or America.

The first method results in water quality concentrations so low that over 95 percent of surface water in the U.S. would fail to meet the criteria. This method also implies that the lifetime risk from current ambient concentrations is approximately three percent, which seems unrealistically high for DDT exposure alone in view of the absence of reported carcinogenic effects in heavily exposed populations. The second method is based on extremely tenuous assumptions, but it does use human data to put an upper limit on the carcinogenic effectiveness of DDT.

*Method 1.* In the Turusov mouse study, the six generations of the lowest dose group (2 ppm) of males had 179 animals with hepatomas out of 354 animals analyzed, whereas in controls 97 out of 328 animals had hepatomas. The data used for the criterion are:

$n_t = 179$
$N_t = 354$
$n_c = 97$
$N_c = 328$
$Le = 104$ weeks.
$le = 104$ weeks.
$d = 2 \times 0.13 = 0.26$ mg/kg/day
$w = 0.030$ kg
$L = 104$ weeks.
$R = 39.000$
$F = 0.0187$ kg/day

With these values the slope parameter is $B_H = 18.055$ (mg/kg/day)$^{-1}$. The result of the calculation is that if fish and water are consumed the water concentration should be less than 0.053 ng/l in order to keep the individual lifetime risk below $10^{-5}$. If only water were consumed ($F = 0$) the corresponding concentration is 20 ng/l.

*Method 2.* There is strong evidence that method 1 overstates the DDT risk, either because the animal experiments overstate the human risk or because most people do not eat fish contaminated to the extent assumed in the model. The basis for stating this is that countries like Israel where the levels of DDT exposure have been high and widespread have experienced no excess cancer incidence as compared to the United States. As an upper limit estimate of cancer risk from DDT exposure, we can make the unsupported assumption that DDT does cause human cancer with some probability which is proportional to the lifetime exposure and we can make the *reductio ad absurdum* argument that cancer incidence in the organ site where the largest excess in incidence occurs in Israel is due solely to DDT, which of course is not true. Taking the nervous

system as a reasonable candidate site for the action of DDT, we can make the following estimate which is intended only to put upper bounds on the carcinogenic effectiveness of DDT.

The high exposure in Israel is reflected in the higher fat levels measured by Wasserman, et al. According to their data the average level in Israel is 16.33 ppm (based on three studies), whereas in the United States it is 9.04 ppm (based on ten studies). Using the following relationship, developed by Hayes, et al. and Durham, et al. between the daily dose I(mg/day), and the concentration, C (ppm) in body fat:

$$\log I = (1/0.7)(\log C - 1.3),$$

the difference in the average daily doses between in Israel and the United States was calculated to be 0.751−0.323=0.428 mg/day. The lifetime incidence of cancer for Israel and New York State is tabulated below from Table 8.3.

| Population (males) | Lifetime incidence (percent) | |
|---|---|---|
| | Nervous System | All sites |
| New York State | 0.5 | 28.8 |
| Israel: | | |
| All Jews | 1.1 | 24.9 |
| Born Israel | 1.3 | 21.4 |
| Born Europe or America | 1.1 | 24.1 |
| Born Africa or Asia | 0.7 | 16.6 |
| Non-Jews | 0.5 | 15.3 |

Relative to New York, the excess lifetime risk of nervous system cancer in migrants to Israel from Europe and America is 1.1−0.5=0.6 percent. This is caused by an excess intake of 0.428 mg/day. Therefore, the intake I resulting in $10^{-5}$ risk is

$$I = (10^{-5}/0.006) \times 0.428 = 7.13 \times 10^{-4} \text{ mg/day}$$

If this intake comes from fish and water, the concentration of water would be:

$$C = 7.13 \times 10^{-4}/(2 + 39{,}000 \times 0.0187) = 0.98 \text{ ng/l}$$

If the intake comes from water alone, the concentration would be:

$$C = \frac{7.13 \times 10^{-4}}{2} = 0.357 \ \mu g/l$$

Therefore, according to method 2, if fish and water are consumed, the water concentration should be less than 0.98 ng/l in order to keep the individual lifetime risk below $10^{-5}$. If only water is consumed, the corresponding concentration is 0.36 μg/l. The equivalent slope factor for method 2 is:

$$B_w = \frac{70 \times 10^{-5}}{2 \times 3.57 \times 10^{-4}} = 0.98 \text{ (mg/kg/day)}^{-1}$$

Method 2 is recommended becuase it gives some basis for avoiding the unrealistically low concentrations imposed by considering only the animal data.

**Hexachlorocyclohexane**

*Criteria Summary*

*Freshwater Aquatic Life.* For lindane the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.21 μg/l as a 24-hour average and the concentration should not exceed 2.9 μg/l at any time.

For freshwater aquatic life, no criterion for a mixture of isomers of BHC can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Saltwater Aquatic Life.* For saltwater aquatic life, no criterion for lindane can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

For saltwater aquatic life, no criterion for a mixture of isomers of BHC can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* For the maximum protection of human health from the potential carcinogenic effects of exposure to α-HCH, β-HCH, and λ-HCH through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of α-HCH, β-HCH, and λ-HCH estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range $10^{-5}$, $10^{-6}$, or $10^{-7}$, with corresponding criteria as follows:

| Isomer | Criteria (ng/l) at the following risk levels | | |
|---|---|---|---|
| | $10^{-5}$ | $10^{-6}$ | $10^{-7}$ |
| α-HCH | 16 | 1.6 | 0.16 |
| β-HCH | 28 | 2.8 | 0.28 |
| γ-HCH | 54 | 5.4 | 0.54 |
| ι-HCH | 21 | 2.1 | 0.21 |

There is insufficient data to establish criteria for the δ and ε isomers of HCH.

*Basis for the Criteria*

*Freshwater Aquatic Life.* The maximum concentration of lindane is the Final Acute Value of 2.9 μg/l and the 24-hour average concentration is the Final Chronic Value of 0.21 μg/l. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

No freshwater criterion can be derived for a mixture of isomers of BHC using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

*Summary of Available Data.* The concentrations below have been rounded to two significant figures.

*Lindane*

Final Fish Acute Value=6.9 μg/l
Final Invertebrate Acute Value=2.9 μg/l
Final Acute Value=2.9 μg/l
Final Fish Chronic Value=2.2 μg/l
Final Invertebrate Chronic Value=1.3 μg/l
Final Plant Value=1,000 μg/l
Residue Limited Toxicant Concentration=0.21 μg/l
Final Chronic Value=0.21 μg/l
0.44×Final Acute Value=1.3 μg/l

*BHC*

Final Fish Acute Value=740 μg/l
Final Invertebrate Acute Value=not available
Final Acute Value=740 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=1,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=1,000 μg/l
0.44×Final Acute Value=330 μg/l

*Saltwater Aquatic Life.* No saltwater criterion can be derived for lindane using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

No saltwater criterion can be derived for a mixture of isomers of BHC using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

**Summary of Available Data**

*Lindane*

Final Fish Acute Value=6.2 μg/l
Final Invertebrate Acute Value=0.076 μg/l
Final Acute Value=0.076 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=1,000 μg/l
Residue Limited Toxicant Concentration=not available
Final Chronic Value=1,000 μg/l
0.44×Final Acute Value=0.033 μg/l

*BHC*

Final Fish Acute Value=23 μg/l
Final Invertebrate Acute Value=0.34 μg/l
Final Acute Value=0.34 μg/l
Final Fish Chronic Value=not available

Final Invertebrate Chronic Value=not available
Final Plant Value=not available
Residue Limited Toxicant Concentration=0.27 μg/l
Final Chronic Value=0.27μg/l
0.44×Final Acute Value=0.015 μg/l

*Human Health.* The animal carcinogenicity data from Ito, et al. 1976, Goto, et al. 1972, Thorpe and Walker, 1973, and Nagasaki, et al. 1972a have been used to develop water quality criteria for α, β, λ, and technical HCH, respectively. These criteria have been developed by the Carcinogen Assessment Group of EPA. The assessment is given in the criterion document.

Under the Consent Decree in NRDC vs. Train, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." α-HCH, β-HCH, γ-HCH and t-HCH are suspected of being human carcinogens. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration of α-HCH, β-HCH and t-HCH in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in some cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of α-HCH, β-HCH, γ-HCH and t-HCH corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria at an interim target risk level of $10^{-5}$, $10^{-6}$, or $10^{-7}$ as shown in the tables below.

| Exposure assumptions (per day) | Risk levels and Corresponding criteria[1] | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ (ng/l) | $10^{-6}$ (ng/l) | $10^{-5}$ (ng/l) |
| **α-HCH** | | | | |
| 2 liters of drinking water and consumption of 18.7 grams fish and shellfish[2] | 0 | 0.16 | 1.6 | 16 |
| Consumption of fish and shellfish only | 0 | 0.18 | 1.8 | 18 |
| **β-HCH** | | | | |
| 2 liters of drinking water and consumption of 18.7 grams fish and shellfish[2] | 0 | 0.26 | 2.6 | 26 |
| Consumption of fish and shellfish only | 0 | 0.32 | 3.2 | 32 |
| **γ-HCH** | | | | |
| 2 liters of drinking water and consumption of 18.7 grams fish and shellfish[2] | 0 | 0.54 | 5.4 | 54 |
| Consumption of fish and shellfish only | 0 | 0.61 | 6.1 | 61 |
| **t-HCH** | | | | |
| 2 liters of drinking water and consumption of 18.7 grams fish and shellfish[2] | 0 | 0.21 | 2.1 | 21 |
| Consumption of fish and shellfish only | 0 | 0.24 | 2.4 | 24 |

[1] Calculated by applying a modified "one-hit" extrapolation model described in the FR 15926, 1979. Appropriate bioassay data used in the calculation of the model are presented in the summary of pertinent data. Since the extrapolation model is linear at low doses, the additional lifetime risk is directly proportional to the water concentration. Therefore, water concentrations corresponding to other risk levels can be derived by multiplying or dividing one of the risk levels and corresponding water concentrations shown in the table by factors such as 10, 100, 1000, and so forth.

[2] Approximately 88 percent of the α-HCH, β-HCH, γ-HCH and t-HCH exposure results from the consumption of aquatic organisms which exhibit an average bioconcentration potential of 780 fold. The remaining 12 percent of α-HCH, β-HCH, γ-HCH and t-HCH exposure results from drinking water.

Concentration levels were derived assuming a lifetime exposure to various amounts of HCH (1) occurring from the consumption of both drinking water and aquatic life grown in waters containing the corresponding HCH concentrations and, (2) occurring solely from consumption of aquatic life grown in the waters containing the corresponding HCH concentrations. Although total exposure information for HCH is discussed and an estimate of the contributions from other sources of exposure can be made, these data will not be factored into ambient water quality criteria formulation until additional analyses can be made. The criteria presented, therefore, assume an incremental risk from ambient water exposure only.

Water quality criteria for the δ and ε isomers of HCH have not been established because of insufficient data. These isomers have not been detected in the environment, however, and would not appear to be a health risk. In summary, the recommended water quality criteria for hexachlorocyclohexane is as follows:

*Form and Criteria*

α isomer 16 ng/l*
β isomer 28 ng/l*
α isomer 54 ng/l*
δ isomer not established—insufficient data
ε isomer not established—insufficient data
technical 21 ng/l

*Summary of Pertinent Data.* The water quality criterion of alpha-hexachlorocyclohexane is derived from the oncogenic effects observed in the liver of male DDY mice fed 500 ppm alpha-HCH in the diet (Ito, et al. 1976). The time-weighted average dose of 65 mg/kg/day was given in the feed for 24 weeks. The liver tumor incidence was 0/18 and 20/20 in the control and treated groups, respectively. Assuming a fish bioconcentration factor of 780, the criterion is calculated from the following parameters:

$n_t$=20 (used 19.5 for calculation)
$N_t$=20
$n_c$=0
$N_c$=18
le=24 weeks
Le=90 weeks
d=500 ppm×0.13=65 mg/kg/day
R=780
L=90 weeks
w=0.0357 kg
F=0.0187 kg/day

Based on these parameters, the one-hit slope, $B_H$, is 2.6637. The resulting water concentration of alpha-hexachlorocyclohexane calculated to keep the individual lifetime cancer risk below $10^{-5}$ is 16 nanograms per liter.

The water quality criterion for beta-hexachlorocyclohexane is derived from the oncogenic effects observed in the liver of male ICR–JCL mice fed 600 ppm beta-HCH in the diet (Goto, et al. 1972). The time weighted average dose of 78 mg/kg/day was given in the feed for 26 weeks. The liver tumor incidence was 0/10 and 10/10 in the control and treated groups, respectively. Assuming a fish bioconcentration factor 780, the criterion is calculated from the following parameters:

$n_t$=10 (used 9.5 for calculation)
$N_t$=10
$n_c$=0
$N_c$=10
le=26 weeks
Le=90 weeks
d=600 ppm×0.13=78 mg/kg/day
R=780
L=90 weeks
w=0.0475 kg
F=0.0187 kg/day

*At a risk level of one in 100,000.

Based on these parameters, the one-hit slope, BH, is 1.5129. The resulting water concentration of beta-hexachlorocyclohexane calculated to keep the individual lifetime cancer risk below $10^{-5}$ is 28 nanograms per liter.

The water quality criterion for gamma-hexachlorocyclohexane is derived from the oncogenic effects observed in the liver of male CF1 mice fed 400 ppm gamma-HCH in the diet (Thorpe and Walker, 1973). The time-weighted average dose of 52 mg/kg/day was given in the feed for 110 weeks. The liver tumor incidence was 11/45 and 27/28 in the control and treated groups, respectively. Assuming a fish bioconcentration factor of 780, the criterion is calculated from the following parameters:

$n_t = 27$
$N_t = 28$
$n_c = 11$
$N_c = 45$
$l_e = 110$ weeks
$L_e = 110$ weeks
$d = 400$ ppm $\times 0.13 = 52$ mg/kg/day
$R = 780$
$L = 110$ weeks
$w = 0.030$ kg
$F = 0.0187$ kg/day

Based on these parameters, the one-hit slope, $B_H$, is $7.7844 \times 10^{-1}$. The resulting concentration of gamma-hexachlorocyclohexane calculated to keep the individual lifetime cancer risk below $10^{-5}$ is 54 nanograms per liter.

The water quality criterion for technical hexachlorocyclohexane is derived from the oncogenic effects observed in the liver of male dd mice fed 660 ppm technical HCH in the diet (Nagasaki, et al. 1972a). The time-weighted average dose of 85.8 mg/kg/day was given in the feed for 24 weeks. The liver tumor incidence was 0/14 and 20/20 in the control and treated groups, respectively. Assuming a fish bioconcentration factor of 780, the criterion is calculated from the following parameters:

$n_t = 20$ (used 19.5 for calculation)
$N_t = 20$
$n_c = 0$
$N_c = 14$
$l_e = 24$ weeks
$L_e = 90$ weeks
$d = 660$ ppm $\times 0.13 = 85.8$ mg/kg/day
$R = 780$
$L = 90$ weeks
$w = 0.0364$ kg
$F = 0.0187$ kg/day

Based on these parameters, the one-hit slope, $B_H$ is 2.0050. The resulting water concentration of technical hexachlorocyclohexane calculated to keep the individual lifetime cancer risk below $10^{-5}$ is 21 nanograms per liter.

## Mercury

### *Criteria Summary*

*Freshwater Aquatic Life.* The data base for freshwater aquatic life and inorganic mercury is insufficient to allow use of the Guidelines. The following recommendation is inferred from toxicity data for saltwater organisms.

For inorganic mercury the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 0.064 μg/l as a 24-hour average and the concentration should not exceed 3.2 μg/l as any time.

For methylmercury the criterion to protect freshwater aquatic life as derived using the Guidelines is 0.016 μg/l as a 24-hour average and the concentration should not exceed 8.8 μg/l at any time.

*Saltwater Aquatic Life.* For inorganic mercury the criterion to protect saltwater aquatic life as derived using the Guidelines is 0.19 μg/l as a 24-hour average and the concentration should not exceed 1.0 μg/l at any time.

The data base for saltwater aquatic life and methylmercury is insufficient to allow use of the Guidelines. The following recommendation is inferred from toxicity data for freshwater organisms.

For methylmercury the criterion to protect saltwater aquatic life as derived using procedures other than the Guidelines is 0.025 μg/l as a 24-hour average and the concentration should not exceed 2.6 μg/l at any time.

*Human Health.* For the protection of human health from the toxic properties of mercury ingested through water and through contaminated aquatic organisms the ambient water criterion is determined to be 0.2 μg/l.

### *Basis for the Criteria*

*Freshwater Aquatic Life.* The maximum concentration of inorganic mercury is the Final Acute Value of 3.2 μg/l which is based on the more acutely sensitive invertebrate organisms. The 24-hour average concentration is 0.064 μg/l and is based on an estimated Residue Limited Toxicant Concentration. No important adverse effects on freshwater organisms of inorganic mercury have been reported to be caused by concentrations lower than the 24-hour average concentration.

The maximum concentration of methylmercury is the Final Acute Value of 8.8 μg/l and the 24-hour average concentration is the Residue Limited Toxicant Concentration of 0.016 μg/l. No important adverse effects on freshwater aquatic life have been reported to be caused by concentrations lower than the 24-hour average concentration.

*Summary of Available Data.* The concentrations herein are expressed as mercury. The concentrations below have been rounded to two significant figures.

*Inorganic Mercury*

Final Fish Acute Value = 38 μg/l
Final Invertebrate Acute Value = 3.2 μg/l
Final Acute Value = 3.2 μg/l
Final Fish Chronic Value = not available
Final Invertebrate Chronic Value = 0.44 μg/l
Final Plant Value = 60 μg/l
Residue Limited Toxicant Concentration = 0.064 μg/l
Final Chronic Value = 0.064 μg/l
0.44 × Final Acute Value = 1.4 μg/l

*Methylmercury*

Final Fish Acute Value = 8.8 μg/l
Final Invertebrate Acute Value = not available
Final Acute Value = 8.8 μg/l
Final Fish Chronic Value = 0.078 μg/l
Final Invertebrate Chronic Value = 0.20 μg/l
Final Plant Value = greater than 2.4 μg/l, less than 4.8 μg/l
Residue Limited Toxicant Concentration = 0.016 μg/l
Final Chronic Value = 0.016 μg/l
0.44 × Final Acute Value = 3.9 μg/l

*Saltwater Aquatic Life.* The maximum concentration of inorganic mercury is the Final Acute Value of 1.0 μg/l which is based on the more acutely sensitive invertebrate species. The 24-hour average concentration is the Residue Limited Toxicant Concentration of 0.19 μg/l. No important adverse effects have been reported to be caused by concentrations lower than the 24-hour average concentration.

No saltwater criterion can be derived or methylmercury using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available. However, results obtained with methylmercury and freshwater organisms indicate how a criterion may be estimated.

For methylmercury and freshwater organisms the Residue Limited Toxicant Concentration is lower than either the Final Fish or Final Invertebrate Chronic Value. Therefore, it seems reasonable to estimate a criterion for methylmercury and saltwater organisms using the Residue Limited Toxicant Concentration.

The maximum concentration of methylmercury is the Final Acute Value of 2.6 μg/l and the 24-hour average concentration is the Residue Limited Toxicant Concentration of 0.025 μg/l.

*Summary of Available Data.* The concentration herein are expressed as mercury. The concentrations below have been rounded to two significant figures.

*Inorganic Mercury*

Final Fish Acute Value=190 μg/l
Final Invertebrate Acute Value=1.0 μg/l
Final Acute Value=1.0 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=1.2 μg/l
Final Plant Value=1.0 μg/l
Residue Limited Toxicant Concentration=0.19 μg/l
Final Chronic Value=0.19 μg/l
0.44 × Final Acute Value=0.44 μg/l

*Methylmercury*

Final Fish Acute Value=not available
Final Invertebrate Acute Value=2.6 μg/l
Final Acute Value=2.6 μg/l
Final Fish Chronic Value=not available
Final Invertebrate Chronic Value=not available
Final Plant Value=100 μg/l
Residue Limited Toxicant Concentration=0.025 μg/l
Final Chronic Value=0.025 μg/l
0.44 × Final Acute Value=1.1 μg/l

*Human Health.* From a health effects perspective and recognition of exposure potential the organo mercury compounds are the most important especially methyl mercury. However, inorganic compounds of mercury should also be recognized because of their toxicity potential but perhaps more importantly because with alkylation from environmentally present biological systems the inorganic mercury can be converted to methyl and dimethyl mercury.

The approach that has been adopted by this criterion document involves the following steps: (1) Identify those organs or tissues most sensitive to damage by the different chemical and physical forms of mercury, damage being defined as an effect that adversely changes normal function or diminishes an individual's reserve capacity to deal with harmful agents or diseases; (2) determine the lowest body burden known to be associated with functional damage in man and, if possible, determine the highest body burden tolerated by man; (3) estimate the potential human intake from ingesting water and eating contaminated fish products; and (4) estimate the effect on body burden of mercury by establishing a criterion for mercury in ambient water based on human health effects.

Table 1, taken from the review by the World Health Organization expert group (1976), indicates long-term daily intakes of methyl mercury which relates to the earliest effect on the central nervous system. This system is more sensitive to damage from methyl mercury than other functional systems in the human body. The conclusions represented in Table 1 were recently endorsed by the National Academy of Sciences (1978).

Evidence reviewed in the criterion document is essentially the same as the evidence reviewed by the WHO group with regard to adult exposures to methyl mercury.

**Table 1.—The Concentrations of Total Mercury in Indicator Media and the Equivalent Long-Term Daily Intake of Mercury as Methyl Mercury Associated With the Earliest Effects in the Most Sensitive Group in the Adult Population [a] [b] [c]**

| Concentrations in indicator media | | Equivalent long-term daily intake (μg/kg body weight) |
|---|---|---|
| Blood (μg/100 ml) | Hair (μg/g) | |
| 20–50 | 50–125 | 3–7 |

[a] The risk of the earliest effects can be expected to be between 3 to 8%.
[b] The table should not be considered independently of the text.
[c] This table is adapted from Table 6 in WHO, 1976.

Effects on the adult nervous system have been estimated to occur at blood concentrations in the range of 200 to 500 ng Hg/ml, corresponding to a long-term daily intake of methyl mercury in the diet of 3 to 7 μg/kg body weight. The risk of effects at this intake level is probably less than eight percent (1 in 12 chances).

Since the WHO (1976) criteria document was written, new evidence has been documented. As reported in the criterion document, females who had experienced maximum hair concentrations during pregnancy in the range of 99 to 384 μg Hg/g had a high probability of having children liable to retarded development. Unfortunately, the population size was too small to establish a lower limit to effects of prenatal exposure. A hair concentration of 99 μg/g is equivalent to a blood concentration of about 400 ng Hg/ml.

The most recent information on effect of mercury on human health has come from the study of the Iraq outbreak of 1971–1972. The follow-up of the cases of prenatal exposure is still in progress. As noted by the National Academy of Sciences (1978), "continued careful evaluation of this very important cohort of pre-natally exposed individuals will provide the most sensitive assessment of human methylmercury toxicity."

Thus, at this stage of knowledge of the dose-effect relationship of mercury in man, it appears that the earliest detected effects in man are at blood concentrations between 200 and 500 ng Hg/ml, for both pre- and post-natal exposures. Blood concentrations of methyl mercury correspond to body burdens in the range of 30 to 50 mg Hg/70 kg body weight, and to long-term daily intakes in the range of 200 to 500 μg Hg/70 kg.

Mercury intake from drinking water, according to data reviewed in the document, is less than 1 μg Hg/day, and is considerably less than the diet portion (Table 2). Assuming that the concentration of methyl mercury in all samples of drinking water is at the current U.S. EPA standard of 2 μg Hg/l, the maximum daily intake would only be 4 μg Hg, assuming 2 liters of drinking water are consumed per person each day. This maximum intake would amount to only about one to two percent of the minimum toxic intake given in Table 2. Thus, from the toxicological standpoint, exposure to mercury via drinking water only would be negligible.

The ingestion of water has been assumed to be the main pathway of direct intake of mercury from water. The transport of mercury through skin is another possible route of intake. Indirect transfer of mercury from water to man is much more important than transfer from direct routes. This conclusion is based on the assumption that fish bioaccumulate a significant amount of methyl mercury from water. In theory, it should be possible to calculate the maximum concentration of methyl mercury in water which would assure that concentrations in edible tissues of fish do not exceed the Food and Drug Administration Guidelines of 1.0 μgHg/g fresh tissue. Thus, if the bioaccumulation factor is known for each species of edible fish, it is arithmetically simple to estimate the maximum concentration of methyl mercury in water. For example, the U.S. EPA (1978) calculated bioconcentration factors (concentration in fish/concentration in water) for methyl mercury compounds based on literature reports. These factors are for edible fish species: 4,525 to 8,376 for rainbow trout *Salmo gardineri*, 20,000 for brook trout *Salvelinus fontinalis*, and 900 to 1,640 for clams *Anodanta grandis, Lampsitis radiata, Lasmigona complanta.* Thus, if the maximum bioaccumulation factor of 20,000 is adopted, the maximum concentration of methyl mercury in freshwater that would prevent fish from exceeding the current FDA guideline would be 0.05 μg/l.

**Table 2.—Estimate of Average and Maximum Daily Intakes of Mercury by the "70 kg Standard Adult" in the U.S. Population [a]**

| Media | Mercury intake μg/day/70 kg | | Predominate form |
|---|---|---|---|
| | Average | Maximum [b] | |
| Air | 0.3 | 0.8 | $Hg^{o}$ |
| Water | 0.1 | 0.4 | $Hg^{++}$ |
| Food | 3.0 | 5.0 | $CH_3Hg^{+}$ |

[a] For details on the calculation of these numbers, see the Exposure section of this document.
[b] These are approximate figures indicating that 95 percent of the population have intakes less than these figures. Occupational exposures are not included.

Unfortunately, both practical and theoretical difficulties thwart any accurate calculation. First, quantitative information is inadequate with regard to the role of direct uptake from water versus accumulation from food chains as contributors to the total amount of methyl mercury in fish. Differences may be expected between fish at lower and upper ends of the food chain. Second, the accumulation factors for methyl mercury uptake by fish are only known for few species. Third, the concentration of methyl mercury in water is probably a variable fraction of total mercury in water. The proportion of methyl to total mercury will probably vary in different bodies of water, being influenced by such factors as water pH, degree of oxygenation, the amount of biota and the sedimentary concentrations of mercury. Fourth, in most cases, the concentration of methyl mercury in water will be so low as to defy accurate measurement even by the most modern technology.

When more information is available on the behavior of mercury in aquatic environments, it might be possible to calculate a reliable criterion based on acceptable concentrations of mercury in fish. In the meantime, a more pragmatic approach will have to be used. The discharge of mercury into bodies of water must be carefully controlled. Those bodies of freshwater supporting edible fish with mercury concentrations above the acceptable levels will have to be identified, and anthropogenic discharge of mercury curtailed. It is also possible that non-anthropogenic sources are predominant (for example, in ocean waters) so that control is not possible. This empirical approach, although the only one available, is unsatisfactory as it allows mainly after-the-fact corrections. Development of procedures for estimating maximum safe concentrations of mercury in ambient water that will prevent unacceptable bioaccumulation of methyl mercury by fish is clearly desirable.

*Methyl Mercury.* Two approaches could be used to derive a criterion for methyl mercury. One approach is to use the existing U.S. drinking water standard of 2 μg/l and the typical water quality exposure assumptions (2 l water/day, 0.0187 kg fish products/day) along with an estimated fish/shellfish bioconcentration factor of 6,200 to calculate a potential uptake. This can then be compared to the Lowest Observable Effect Level (LOEL) to determine the range of safety. A second approach is to use the LOEL as a basis for establishing an acceptable daily intake (ADI) and calculate a criterion level using the typical water quality assumptions.

Given: fish/shellfish consumption = 0.0187 kg fish/person/day
bioconcentration factor for methyl mercury = 6,200 = $\frac{\text{mg Hg/kg fish}}{\text{mg Hg/l water}}$
Water consumption = 2 l/person/day
(1) Assume
criterion = 2 μg/l
Human exposure = 2 l/day + (6,200 × 0.0187)
= 2 (2 + 115.9)
= 235.8 μg/day

Recognizing that the LOEL range is 200 to 500 μg Hg/day, we could hypothesize that there is little or no margin of safety at the 2 μg/l criterion level especially where realizing that dietary sources other than fish products may be contributing to the body burden.

(2) Derive ADI using typical water quality exposure and LOEL
LOEL range = 200–500 μg Hg/day
Use 200 μg Hg/day to assure marginal safety
ADI = 200 μg/day
= C 2 l/day + (6,200 × 0.0187)
200 = C (2 + 115.9)
200/117.9 = C
1.7 μg/l = C

According to the National Academy of Science (1977) an uncertainty factor of ten can be applied to the ADI as the 200 to 500 data results from studies on prolonged ingestion by man, with no indication of carcinogenicity.

200/10 = C (2 + 115.9)
0.17 μg/l = C
0.2 μg/l ~ C

Whereas, approach No. 1 has an estimated narrow margin of safety if any and given that LOEL's do exist it is reasonable to focus on the ADI based criterion with an uncertainty factor as the preferred basis for establishing a criterion.

**Polynuclear Aromatic Hydrocarbons**

*Criteria Summary*

*Freshwater Aquatic Life.* For freshwater aquatic life, no criterion for any polynuclear aromatic hydrocarbon can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Saltwater Aquatic Life.* For saltwater aquatic life, no criterion for any polynuclear aromatic hydrocarbon can be derived using the Guidelines, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* For the maximum protection of human health from the potential carcinogenic effects of exposure to polynuclear aromatic hydrocarbons (PAH) through ingestion of water and contaminated aquatic organisms, the ambient water concentration is zero. Concentrations of PAH estimated to result in additional lifetime cancer risks ranging from no additional risk to an additional risk of 1 in 100,000 are presented in the Criterion document. The Agency is considering setting criteria at an interim target risk level in the range of $10^{-5}$, $10^{-6}$, or $10^{-7}$ with corresponding criteria of 9.7 ng/l, 0.97 ng/l and 0.097 ng/l, respectively.

*Basis for the Criteria*

*Freshwater Aquatic Life.* No freshwater criterion can be derived for any polynuclear aromatic hydrocarbon using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

*Saltwater Aquatic Life.* No saltwater criterion can be derived for any polynuclear aromatic hydrocarbon using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available, and there are insufficient data to estimate a criterion using other procedures.

*Human Health.* The presently available data base is inadequate to support the derivation of individual criteria for each of the PAH as specified under the Consent Decree. This problem arises primarily from the diversity of test systems and bioassay conditions employed for determining carcinogenic potential of individual PAH in experimental animals. Furthermore, it is not possible to estimate the intake via water of individual PAH except for those compounds which have been selected by the World Health Organization for environmental monitoring. Therefore, an approach to criterion development is adopted in this report with the objective of deriving a single criterion to encompass the entire PAH class. This approach is attractive in that it recognizes the fact that environmental exposure to PAH invariably occur by contact with complex, undefined, PAH mixtures.

The attempt to develop a drinking water criterion for PAH as a class is hindered by several gaps in the scientific data base:

(1) The PAH class is composed of numerous compounds having diverse biological effects and varying carcinogenic potential. A "representative" PAH mixture, has not been defined.

(2) The common practice of using data derived from studies with BaP to make generalizations concerning the effects of environmental PAH may not be scientifically sound.

(3) No chronic animal toxicity studies exist involving oral exposure to PAH mixtures.

(4) No direct human data exist concerning the effects of exposure to defined PAH mixtures.

However, assuming that the development of a criterion must proceed despite these obstacles, certain approaches may be taken to circumvent deficiencies in the data base. The choice of an appropriate animal bioassay from which to derive data for application to the linear non-threshold model for human cancer risk assessment should be guided by several considerations. Primary emphasis must be placed on appropriate animal studies which: (1) Include sufficient numbers of animals for statistically reliable results; (2) involve long-term low-level exposures to PAH; (3) include a proper control group; and (4) achieve positive dose-related carcinogenic response.

Because there are no studies available regarding chronic oral exposure to PAH mixtures, it is necessary to derive a criterion based upon data involving exposure to a single compound. Even when considering single chemicals, almost no studies are available which involved oral exposure at more than one dose level to a reasonable number of animals. Two studies have been selected, one involving BaP ingestion (Rigdon and Neal, 1967) and one involving DBA ingestion (Snell and Stewart, 1962). Both compounds are recognized as animal carcinogens, and both are known to be environmental contaminants to which humans are exposed.

In the strictest sense it can be argued that a criterion for a chemical class derived from experiments involving a single component of that class is invalid. On the other hand, selection of those components (e.g., BaP and DBA) which are among the more potent carcinogens in the PAH class should lead to a conservative criterion approach. It must be assumed that interactions among the various PAH components resulting in either an enhancement or inhibition of biological effect will cancel each other out in the environment. Presently, there is no way to quantitate the potential human health risks incurred by the interaction of PAH, either among themselves or with other agents (e.g., tumor initiators, promoters, inhibitors) in the environment. In addition, it is known that PAH commonly produce tumors at the site of contact (i.e., forestomach tumors by oral exposure to BaP; lung tumors by intractacheal administration; skin tumors by dermal application). Thus, consideration of the extent of absorption may not always be necessary in the case of carcinogenic PAH, and will in fact result in underestimation of actual risk if only distant target sites are considered. Calculations of water quality criteria for PAH based upon bioassay data for BaP and DBA are presented in the summary of pertinent data.

The water quality criteria for BaP and DBA derived using the linear non-threshold model as described in the Appendix are 27.5 ng/l and 43 ng/l, respectively. For the sake of comparison, a water quality criterion for DBA was calculated using the procedure developed by Mantel and Bryan (1961). As opposed to the linear non-threshold model, which is logistic and defines acceptable risk as 1/100,000, the Mantel and Bryan (1961) model is probalistic and defines acceptable risk as 1/100,000,000. Furthermore, the Mantel and Bryan (1961) is concerned with the maximum tumor incidence in treated animals at the 99 percent assurance level. Using the Mantel and Bryan (1961) approach with DBA, the resultant water quality criterion is 13.3 ng/l.

Under the Consent Decree in NRDC v. Train, criteria are to state "recommended maximum permissible concentrations (including where appropriate, zero) consistent with the protection of aquatic organisms, human health, and recreational activities." BaP and DBA are known animal carcinogens. Because there is no recognized safe concentration for a human carcinogen, the recommended concentration in water for maximum protection of human health is zero.

Because attaining a zero concentration level may be infeasible in cases and in order to assist the Agency and States in the possible future development of water quality regulations, the concentrations of BaP and DBA corresponding to several incremental lifetime cancer risk levels have been estimated. A cancer risk level provides an estimate of the additional incidence of cancer that may be expected in an exposed population. A risk of $10^{-5}$ for example, indicates a probability of one additional case of cancer for every 100,000 people exposed, a risk of $10^{-6}$ indicates one additional case of cancer for every million people exposed, and so forth.

PAH are widely distributed in the environment as evidenced by their detection in sediments, soils, air, surface waters, and plant and animal tissues. The ecological impact of these chemicals, however, is uncertain. Numerous studies show that despite their high lipid solubility, PAHs show little tendency for bioaccumulation in the fatty tissues of animals or man. This observation is not unexpected, in light of convincing evidence to show that PAH are rapidly and extensively metabolized.

Lu, et al. (1977) have published the only available study regarding the bioconcentration and biomagnification of a PAH in model ecosystem environments. They reported that the bioconcentration of BaP, expressed as concentration in mosquitofish/concentration in water was zero. This was apparently due to the fact that the fish metabolized the BaP about as rapidly as it was absorbed. On the other hand, in a 33 day terrestrial-aquatic model ecosystem study, BaP showed a small degree of biomagnification which probably resulted from food chain transfer. In this case the biomagnification factor for mosquitofish was 30. Based on the results of Lu, et al. (1977) a bioconcentration (BCF) factor of 30 was employed for the purpose of calculating a water quality criterion. In contrast, as can be noted in the criterion document, the Cancer Assessment Group applied the BCF of 6800, a value derived from octaonol-water partition coefficients, in its calculation of the water quality criterion for PAH.

In the **Federal Register** notice of availability of draft ambient water quality criteria, EPA stated that it is considering setting criteria for BaP and DBA at an interim target risk level of $10^{-5}$, $10^{-6}$ or $10^{-7}$ as shown in the table below.

| Exposure assumptions (per day) | Risk levels and corresponding criteria [1] | | | |
|---|---|---|---|---|
| | 0 | $10^{-7}$ (ng/l) | $10^{-6}$ (ng/l) | $10^{-5}$ (ng/l) |
| **BaP** | | | | |
| 2 liters of drinking water and consumption of 18.7 grams fish and shellfish [2] | | 0.097 | 0.97 | 9.7 |
| Consumption of fish and shellfish only | | 0.44 | 4.45 | 44.46 |
| **DBA** | | | | |
| 2 liters of drinking water and consumption of 18.7 grams fish and shellfish [2] | 0 | 0.043 | 0.43 | 4.30 |
| Consumption of fish and shellfish only | | 0.196 | 1.96 | 19.63 |

*Summary of Pertinent Data.* The water quality criterion for PAH is based on the experiment reported by Neal and Rigdon (1967) in which benzo(a)-pyrene at doses ranging between 1 and 250 ppm in the diet was fed to strain CFW mice for approximately 110 days. Stomach tumors, which were mostly squamous cell papillomas but some carcinomas, appeared with an incidence statistically higher than controls at doses of 45 ppm and above. At 45 ppm the incidence in controls and treated groups was 0/289

and 4/40, respectively. The one-hit model has the following parameters:

$n_t = 4$
$N_t = 40$
$n_c = 0$
$N_c = 289$
Le = 110 days
le = 110 days
d = 45 ppm × 0.13 = 5.85 mg/kg/day
w = 0.034 kg
L = 78 weeks × 7 days/wk = 546 days
R = 30
F = .0187 kg/day

With these values, the one-hit slope parameter is $B_H = 28.020\ (mg/kg/day)^{-1}$.

The result is that the water concentration of BaP should be less than 9.7 nanograms per liter in order to keep the individual lifetime risk below $10^{-5}$. On the conservative assumptions that all carcinogenic PAH compounds are as potent as BaP, that the effect of a mixture of carcinogenic PAH compounds depends on the sum of their concentrations, and that the non-carcinogenic PAH compounds have no effect on the response of the carcinogenic PAH, it follows that the sum of the concentration of all carcinogenic PAH compounds should be less than 9.7 nanograms per liter in order to keep the lifetime risk less than $10^{-5}$.

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